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Pandemic influenza: an analysis of the spread of influenza in Kitchener, October 1918.

By

Niall Philip Alan Sean Johnson
Bachelor of Science (Honours), Macquarie University, 1991.

THESIS
Submitted to the Department of Geography
in partial fulfilment of the requirements
for the Master of Arts degree
Wilfrid Laurier University
1993

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Canada

Only a little more
I have to write
Then I'll give o'er
and bid the world Good-night.

Robert Herrick "His Poetry His Pillar"

Then, rising with Aurora's light
The Muse invoked, sit down to write;
Blot out, correct, insert, refine,
Enlarge, diminish, interline.

Jonathon Swift "On Poetry" (1733)

Abstract

Influenza remains one of the major killers in modern society. In addition to the mortality it causes, it exacts a huge medical, social and economic toll. Due to its propensity to undergo change, through antigenic drift and shift, this disease continues to torment humankind. These changes are the driving force that enables influenza to periodically become epidemic and pandemic. The study of past pandemics thus may provide useful insight to combat the disease now and in the future.

The 1918-19 pandemic is one of the three worst outbreaks of disease in recorded history, with only the Justinian plagues and the Black Death claiming more victims. This pandemic has largely disappeared from our collective memory. In this study the pandemic is examined at various scales: global, continental, national and local. Examination of the origins and spread of the disease, the mortality, particularly variations in that mortality with respect to age, sex and socio-economic conditions are made. Particular attention is paid to the experience of Kitchener, Ontario where the spatial aspects of the disease, along with other factors, are scrutinised. Using primary source data the Kitchener experience of this pandemic is examined and the passage of the disease is mapped and analysed.

This study establishes that Kitchener indeed suffered greatly from the 1918 influenza pandemic, with the crude death rate rising almost sevenfold for the month of October. The pattern of mortality, especially age-sex mortality, was very similar to that found elsewhere in North America. Socio-economic factors appear to have had some influence upon mortality arising from the disease.

The influenza pandemic of 1918 displayed both hierarchical and contagious components to its diffusion. The disease relocated through the national urban hierarchy and then exhibited contagious diffusion at the local level.

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I would also like to thank all of those people, my friends, my colleagues, who have helped me see this thesis to fruition.

Thanks also to Piers for his invaluable assistance in completing the maps, and to Donna for the vital finishing touchs.

Ricki, who has shown me much about friendship and myself.

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Appendix III

Chapter 1. Objectives, methodology, data.

'Ye can call it influenza if ye like,' said Mrs Mackin. There was no influenza in my young days. We called a cold a cold. Arnold Bennett "The Card", chapter 8.

In analysing the passage of the influenza pandemic of 1918 through Kitchener it is necessary to first understand the nature of the disease, how the pandemic struck elsewhere, what other authors have revealed about influenza and the study of historic episodes of infectious disease and a myriad of other factors. Consequently this paper is structured so that much of this material is reviewed and presented before entering into the examination of the Kitchener experience. This opening chapter introduces the problem briefly, considers the geographical philosophies guiding the paper, discusses in some detail the data, the data sources and the problems and limitations of that data and concludes with some comments on the methodology. The following chapter reviews the relevant literature while Chapter 3 provides important information on the disease itself before moving onto a detailed examination of the 1918 pandemic, including the origin and spread of the disease at various scales. Chapter 4 sees

the focus move to the local scale, introducing our study area - the city of Kitchener. This is largely a descriptive chapter documenting how the disease manifested itself in the city, drawing heavily from the primary data and finishing with some examination of the morbidity (illness) and mortality (death) caused. The Kitchener experience described in Chapter 4 is then analysed in Chapter 5. Here the spread of the disease is mapped and analysed, along with examination of the associated spatial variation in mortality with regard to age and socio-economic concerns. Following this analysis are the conclusions and implications of this research (Chapter 6).

Influenza. To most of us an occasional malady that knocks us off our feet for a couple days every few years. Few of us think much of it, it is an inconvenience but no more. Most of us do not consider it a major threat to our continued existence. However, influenza does kill. Influenza can exact a massive toll. A toll of human life and suffering; it is a disease that has a massive, hidden, social and economic cost. Each passage of influenza sees a major portion of the population infected. While only a tiny proportion of these people die, the sheer volume of people infected places a major drain on our medical resources. The loss of production, the cost of vaccines, the time taken to care for sufferers, all represent a diversion of human time and resources. In the United States alone one recent

estimate contended that "By conservative estimates, the cost of influenza averages more than $1 billion each year, and it is likely that the actual cost, were it calculable, would be on the order of $3 to $5 billion per year." (Schoenbaum, 1987:26) Such calculations are by their very nature difficult, as they must take into consideration

> all of the costs attributable to influenza virus infection, including the costs of all medical services used by persons with influenza (physicians' fees, laboratory tests, prescription and non-prescription drugs, hospital charges for acute and extended care, and special equipment, amongst others). These are the direct costs of influenza . . . Yet these direct costs represent only a fraction of the total costs - approximately 20 to 30 percent. (Schoenbaum, 1987:26)

In addition to the direct medical costs are the indirect medical costs and the drain on the economy at large. When one starts to consider such costs, and the estimates tend to be on the conservative side, Schoenbaum claims that for the United States alone influenza 'costs' $3 to $5 billion, and this is for every normal year. The cost of an epidemic, severe outbreaks of the illness that may be localised, or worse yet, a pandemic, a global epidemic usually associated with a new viral subtype, is unthinkable.

Influenza remains among the ten leading causes of death in the United States. Douglas notes that in the United States approximately "500,000 people

have died during influenza outbreaks in the past 20 years, [1967-1987] and more than 40,000 excess deaths have been documented during several recent epidemics. Even in nonepidemic years, as many as 20,000 people may die from influenza" (1987:1) in that one country alone! The Canadian picture is similar in that "it is estimated that influenza has killed about 17,000 Canadians during the past 20 years [1947-1967] but . . . the true figure is probably 30% greater. Similarly it is estimated that influenza was responsible for about 5% of all sickness absences and nearly 3% of all time lost from work because of sickness" (McDonald, 1967:526). The impact of a pandemic is almost unimaginable, both in terms of human suffering and economic loss. The study of past pandemics makes a significant contribution to resolving the "current mysteries about influenza" (Patterson, 1986:xvi) as "accurate knowledge of the past will provide a fuller understanding of the range of behaviour of influenza" (Patterson, 1986:xvi).

The influenza pandemic that swept the globe in 1918 remains among the greatest mortality events in human record. It is claimed that over half the world's population at that time, in excess of a billion people, suffered the ravages of the virus and that at least 20 million of these attacks were fatal! McGinnis notes that "[F]or Canada, the number of sufferers was set at about two million" (1976.1) of

whom at least thirty thousand died. (Pettigrew, 1983:xv) And yet, as McDonald notes, "[I]nfluenza has received rather less attention in Canada than in countries such as the United States and Britain, possibly because the importance of the disease has not been fully appreciated . . ." (1967:522).

Kitchener's experience of this most devastating global scourge is most interesting, largely unrecorded, and a valid topic for detailed study for a number of reasons. One of these is Kitchener as an example of the Spanish influenza experience - a detailed examination of a single city. In a single site we can examine how the disease manifested itself, how mortality was visited upon the people, in terms of its location, its timing and its extent, the public reaction, the actions of the populace, particularly their leaders, what role the political processes played in hindering or helping the disease strike, what influence the social geography of the city had upon the nature of the disease. All of these facets of this manifestation of the Spanish Influenza of 1918 shall be examined here. This study of Kitchener is complemented by, and complementary to, an inspection of how the disease expressed itself at various levels, including global, continental and regional patterns. This study would appear to be a classic case study of disease diffusion. However, it is more than simply a study of diffusion as a mathematical and cartographic exercise. Rather it is a study of one of the

worst pandemics in which not only the spatial aspects of the disease are examined but the human aspects of the pestilence are brought to the fore.

Constraints, including time and extant data, dictate that one cannot produce an exhaustive study of all aspects of Kitchener's experience. However, working within these constraints enough data is available to adequately analyse this subject; further data would only produce marginal results. The limitations of the historical data shall be discussed further in this chapter. This paper will examine the disease at various scales, but it shall be more than this in that it endeavours to consider the disease in the entire physical, social, political and economic context in which it occurred. A medical geography approach lends itself to such a study as Meade *et al.* (1988:3) counsel when they introduce the field thus.

> Medical geography uses the concepts and techniques of the discipline of geography to investigate health-related topics. Subjects are viewed in holistic terms within a variety of cultural systems and a diverse biosphere. Drawing freely from the facts, concepts and techniques of other social, physical and biological sciences, medical geography is an integrative, multistranded subdiscipline that has room within its broad scope for a wide range of specialist contributions.

But why study influenza, particularly a past pandemic? Influenza persists as a major cause of illness and death across the globe and is one of the most important infectious diseases we face. While its mortality rate is relatively low,

its high morbidity make it medically, socially and economically important. As was noted previously, the disease persists as a major global epidemic disease. Furthermore, its propensity for antigenic drifts and/or shifts into strains of varying virulence makes the possibility of future epidemics quite feasible. Patterson, (1986:xv) in introducing his examination of pandemic influenza, asserts that "[R]esearch on past epidemics has both historical and epidemiological significance." Thus, the study of past pandemics can quite possibly provide some insight that may help us combat this disease in its current and possible future forms. A better understanding of the disease, such as how it moves through populations, will stand us in better stead when we again face it.

Introduction to the pandemic

As the Great War rumbled to a halt in the latter half of 1918 the world came under attack from a far more invidious enemy - influenza. The first outbreaks of the disease apparently came in early spring of 1918 with cases reported in China and Japan and also among American, British (including Commonwealth), and French troops in France (Marks and Beatty, 1976). It was these infected troops who were to become such ready vectors for the disease as they were demobilised and returned to their communities around the globe. Pettigrew (1983:4) notes how apt the adage 'after war comes plague' was to prove in 1918-19.

Canada lost almost sixty thousand of its young men during the years of the First World War. Influenza struck with even greater force at home, mercilessly claiming tens of thousands in a few short months. Collier (1974.305) cites the American epidemiologist, Edwin Oakes Jordan, in claiming that total global mortality was 21 642 274, with influenza morbidity attacking more than "a billion people - more than half the world's population" (Collier, 1974:305), while Meade *et al.* note that the "1918-19 pandemic was extraordinarily virulent, with total global mortality estimated at between 20 and 50 million." (1988:239). Schild summarises the passage of the pandemic thus:

> The pandemic occurred in several waves during a period of little over a year. The first, in spring of 1918, was relatively mild, the attack rate being 20-40% in those aged up to 50 years but with a considerably lower rate in the aged. The second wave in the autumn of 1918 was exceptionally severe, producing enormous mortality in the 20-40 year age groups. A less severe wave occurred in early 1919 (Schild, 1977:366).

The spatial aspects of influenza are well-known (Cliff *et al.* 1986, Howe 1977, Patterson 1986, Pyle 1979, Pyle 1980, Pyle 1986, Selby 1982), with diffusion of the disease being of foremost concern to many medical geographers. The pandemic of 1918 in Kitchener provides us with a historical stage on which we can play out our reconstruction of the spread and impact of the disease.

The study of disease, particularly the study of epidemics, as Curson and McCracken, two of the foremost Australian medical geographers, note, "reveals them to be complex and fascinating phenomena with social and spatial implications well worthy of geographical investigation." (1989:11.6) These spatial and social features are the focus of this study. Curson and McCracken continue that "in geographical terms an epidemic represents a convergence in time and space of an agent (and sometimes also a vector) and a susceptible population involving sequences of socio-spatial interaction, action and reaction." It is these sequences that shall be examined here. It is hoped that by examining this particular occurrence at various scales one can develop a greater understanding of the dynamics of the pandemic.

One area of study that has recently surfaced in geography has been "a concern with understanding the distribution of infectious disease in past populations and the nature of the spatial diffusion of historic epidemics." (Curson and McCracken, 1989:11.7) This study then, quite obviously, fits into this recent trend in geographical inquiry.

Philosophical considerations

Before we enter any detailed examination of the case it is necessary to acknowledge the philosophical and methodological approaches taken here as these have a major influence in the execution of the study. The philosophical approach here is rather positivistic. That is, an implicit belief in the verifiable and replicable 'laws' and models of the 'real' world. Our re-construction of the pattern of death is a manifestation of the belief that we can know our universe, identify trends, make generalisations, ascribe laws and predict processes and the resulting patterns. However, one shall also try to utilise the "historical mind" that Harris (1978:285) speaks of, which, combined with Harris' earlier discussion of the "theory and synthesis of historical geography" (1971:147) is rather reminiscent of Capra's (1982) call for more holistic approaches in modern study. These views complement the description of medical geography given by Meade *et al.* (1988:3):

> Subjects are viewed in holistic terms within a variety of cultural systems and a diverse biosphere. Drawing freely from the facts, concepts and techniques of other social, physical and biological sciences, medical geography is an integrative, multistranded subdiscipline .

Thus the disease shall not be considered in isolation, rather I shall attempt to consider the spread of influenza mortality in its fullest context. Emphasis shall be

placed upon the description of the diffusion of the disease through the city and comparison of the Kitchener experience with that encountered elsewhere.

Data sources

In attempting to reconstruct a geography of disease mortality it is vital to identify all possible, probable and even the improbable data sources. However, there exist a myriad of problems facing the medical historical geographer. Here the problems with data are not simply those common to the medical geographer or the medical researcher but they are compounded greatly by the problems of historical data. Firstly one must state what types of data are of interest and of use. Data relating to both mortality (fatalities caused by the disease) and morbidity (illness related to the disease) are invaluable. Obviously any material relating to the influenza pandemic as it passed through the city is of interest, but to be of use in a geographical study of disease then it must give us particular information. In studying mortality what is required is the spatial location of each fatality, the home and work locales for each individual who succumbs. Thus we require the name and address for each case as a minimum data requirement if we are to map them.

Following is a brief discussion of each potential data source in the region [as suggested by Curson and McCracken (1989), Dunn and Baldwin (1983) and

MacNaughton and Wagner (1989)], and the potential or inherent problems with each.

Kitchener Public Library and Waterloo Historical Society

The very first place to look for background information and, possibly, detailed information was the local library. Fortunately this also houses the collection of long-established (1912) Waterloo Historical Society. It was ascertained that this region had indeed suffered from the influenza epidemic of 1918. However, the extent of that suffering was unclear.

Unfortunately the local histories (Conrad 1987, Donohoe 1954, English and McLaughlin 1983, Moyer 1979, Rowell *et al.* 1982, Taylor 1990, Uttley 1937) available at the various libraries around the city (Kitchener Public Library, Waterloo Public Library, Wilfrid Laurier University Library and the University of Waterloo Library) tend to exemplify the maxim that "history is about chaps and geography is about maps" (Wynn, 1990:13) That is, they emphasise the people involved in the development of the city and spend little time discussing processes, the development of the city and events such as the influenza pandemic. Surprisingly very few of the local histories of either the city of Kitchener or the region of Waterloo detail the impact of the influenza epidemic that shook the town. Indeed the pandemic is conspicuous by its almost

non-existence in the local histories. For an event that had such an impact it has gone largely unrecorded.

This situation was repeated at the Waterloo Historical Society collection, where genealogical information seems a favourite topic. Much interesting information is held by the Society and they produce quite engrossing material. However, the influenza epidemic is rarely referred to directly. It occasionally crops up in passing, in a number of documents and in some oral history recordings, (Campbell 1986, Clarke and Clarke 1982, Hagmeier 1981, Taylor 1990, Waterloo Historical Society 1918, 1920) but little of direct relevance to this project has come from their holdings.

However, the Waterloo Historical Society does hold the extremely detailed City of Kitchener Assessment Rolls for the time. These volumes detail each and every property in the five wards of the city, giving information on the name and occupation of the owner/tenant, the value of the land and the buildings, the total assessed value and the taxes assessed. Also held is the Income Tax roll which provides further information about some of the city's residents. This information is invaluable in understanding the socio-economics of the city and the disease's victims.

Waterloo Regional Health Unit

As this is a piece of research into a health problem one potential data source are the records of the local health organisation(s). Waterloo Regional Health Unit was thus approached regarding past health records as Kitchener falls under the jurisdiction of this unit. However, as they are a relatively recent entity (1975) they possessed no records pertaining to the 1918 influenza epidemic, despite having been created by the amalgamation of similar, smaller, units. In 1964 the Galt Health Unit joined with the Waterloo County Health Unit with the cities of Kitchener and Waterloo joining in 1968. 1973 saw the county unit become a "free standing independent Board of Health." (MacNaughton and Wagner, 1989:97) This board was then disbanded in 1975 and the Waterloo Regional Health Unit reported to the Health and Social Services Committee, a standing Committee of Regional Council. Furthermore, they possessed no readily available information as to what may have happened to the records of their predecessors.

City of Kitchener

Weiler's 1988 work suggested that City Clerk's Office of the City of Kitchener as another source of data. However, on visiting it became apparent that the only material that referred to the epidemic was a number of the minutes of the city's Board of Health. Weiler does quote from these, but it had been

hoped that other, more specific, material would be available. But this was not to be so. Other sections of the City of Kitchener bureaucracy, including the cemetery, were consulted to ascertain if any useful material was held anywhere else within the system. Unfortunately these efforts proved fruitless. What City of Kitchener records that have proved of use, beyond the Board of Health minutes, are the Assessment Rolls and Income Tax Roll housed in the Kitchener Public Library.

Kitchener-Waterloo Hospital

From Craig's inventory of Ontario and London hospital records (Craig, 1992) and from MacNaughton and Wagner (1989:46-7) we know that somewhere within the Kitchener-Waterloo Hospital there exist some records from the period. These include Annual Reports, By-Laws, Deeds and Trusts, Operating room registers and the Women's Hospital Auxiliary minutes. Examination of these revealed very little. The impact of the disease is apparent but specifics are scarce.

Kitchener-Waterloo Academy of Medicine

Despite being a relatively recent creation, founded in 1950, it was thought that this local branch of the Ontario Medical Association may be able to shed some light on the situation regarding doctors' records and recollections.

Unfortunately this was not the case; they were unable to produce any material that could shed any light on the pandemic.

Provincial Archives of Ontario

The Ontario archives house a most extensive collection of material in Toronto. However, the information available may not always be of particular, direct, relevance. This was, unfortunately, the case here. While the archives contained useful background or secondary information such as copies of telegraphs requesting provincial assistance, little data existed that would assist in attempting to map the impact of influenza in Kitchener. Other data sources were necessary to obtain this kind of information. However, it should be noted that the archives also contain the Registrar-General's reports and the local Medical Officer's reports collated in the provincial health records. Greater use of this material may have added to this study, and should be considered in future study..

Newspapers

Thus it is the city's newspapers of the time that have provided the bulk of the primary information upon which this work is founded. It had been expected that official statistics of illness and death coming from either local or provincial authorities would provide this foundation. However, it was found that this

information does not, apparently, remain in existence whereas the contemporaneous newspapers had been preserved and contained far more copious data than may have been expected.

Not only do the local newspapers of the time (the *Kitchener Daily Telegraph* and the *News-Record*) chart the progress of the epidemic, right from its earliest stages, they also appear to have recorded every death. It had been thought that the newspapers would have only come upon the 'story' well after the disease had established itself. However, this was not the case, as the disease had been experienced elsewhere, both around the world and in Canada, including the local region. Furthermore, the depth of information on each individual death was somewhat unexpected. It had been thought that simple statements of who had died and when might exist, and then only for some of the deaths. Instead we find relative detailed paragraphs relating the name of the deceased, the date of their death and their address, in most cases.

Thus it is the city's two newspapers that allow us to gain some insight into the impact of influenza on the city. The stories printed tell us not only of the deaths and the suffering in the city but also give us some idea about what people thought, what actions were being taken and the impact of the disease upon the city. They provide us with not only the primary data regarding the mortality

brought about by the illness but also colour our image of the city, they paint a picture of the city and its inhabitants by telling us of the victims, the actions being taken to stem the disease and the like. However, their most important role is to indicate to us who succumbed to the disease in the city. It is the obituaries and tallies of death that interest us the most. These provide us with the temporal information required and, quite often, with the spatial data relating to the home address of the victims. In those cases where the spatial data is missing, the papers not giving the residential address of the deceased, the city directories for 1918 and 1919 (Cairnes 1918, Vernon 1919) and the city's assessment rolls were consulted, and in most instances the required information was located.

Doctor's records

From the newspapers it is known that some eighteen doctors were practising in the city at the time the pandemic struck. Campbell's (1986) inventory of the county's doctors over the years allows one to identify each of those doctors (Appendix V) and then theoretically it may be possible to track down their personal records, journals and writings. However, there is no such extant material for this region. Furthermore, even if they do exist it may not be possible to obtain access to them for various reasons. Nevertheless, this is one avenue that could be explored in any future search for more and more

information with which to re-construct the Kitchener experience. The only material recovered has been a Waterloo Historical Society interview of Dr. J Edwin Hagmeier in which he briefly recalls the influenza pandemic of 1918. (Hagmeier 1981)

Secondary sources

Unfortunately there is a distinct paucity of secondary material. The journals, for example *Ontario History* and the *Canadian Bulletin of the History of Medicine,* that might contain material pertaining to the 1918 pandemic, and particularly in Kitchener, were found to contain no such material. Thus a widespread search for material encompassed many journals from many disciplines with only occasional success. The bulk of this material relates to the pandemic as encountered elsewhere, predominantly in the United States of America. However, it should be noted that the *Canadian Public Health Journal* has published a number of items relating to influenza over the years, and greater use of these should be made in future

Data problems and limitations

Many are the data problems that dog the historical geographer and the medical historian, let alone the medical historical geographer! There are the

problems of historical data, such as existence of any useful information, the problems of interpretation, changes in nomenclature, problems of temporal and cultural separation. Data may have been collected and collated in ways that are not apparent or meaningful to us in the 1990s. Then there are the problems of medical data, including the dubious accuracy of cause of death statements. Combining all of these into a geographical framework brings yet more problems. Is the data spatially organised? Can one re-create the spatial variations of the disease from the available data?

There are many potential problems with historical medical data. Curson and McCracken (1989) discuss a number of these. Problems that were anticipated in this study included not only the likelihood of incomplete, possibly non-existent, official (hospital, death registers, etc.) records but also the likelihood that the extant data may be of little use in attempting to reconstruct the spatial movement of the disease. That is, the data could well have been aggregated (for example, for the whole city) resulting in our having little idea where the disease moved through the city in terms of either mortality or morbidity. Another possibility is inaccurate ascribing of the cause of death, a very common problem in medical data. This is considered unlikely here though as the disease was known and its symptoms recognised. Indeed in times of epidemic there may be a

problem of over-reporting in that influenza may be recorded as cause of death in cases where death was actually the result of something else, e.g. heart attack. However, a major problem with institutional data, such as hospital records or death registries, has been its apparent non-existence.

A major stumbling block for anyone considering past episodes of many diseases, including influenza, is the lack of evidence regarding morbidity. This appears to be particularly true of influenza, as the disease is so common and is often confused with the common cold with many cases going unreported. Where morbidity data is found it is often simply in the form of reported cases over a period. Such information has a number of limitations. Firstly, it is likely to be an underestimation of the true morbidity, and secondly it has no temporal or spatial element - there is no intelligence regarding where or when in the time period the cases occurred. Thus little information relating to morbidity exists, and when it does it tends to be anecdotal. For example, Dr. Hagmeier, in recalling his experience in Kitchener, tells of "another time I was called in to see someone . . . it was in the attic and they had a dozen cots and they've all of them flu . . . there were some very sick people." (Hagmeier, 1981) Furthermore, when pressed he would not even hazard a guess at the morbidity rate.

Mortality is usually the only available data. Death is a yes or no diagnosis and tends to be recorded. In our study deaths were recorded in the local papers, among other places. Thus mortality data must function as an indicator of how the disease moved at various levels and its impact upon the community. Mortality is often used as a surrogate of influenza activity (Fine 1982, Monto 1987, Patterson 1986).

The extant Kitchener data shares many of these difficulties. The tally of deaths is incomplete. There are gaps in the records of the residential addresses of the victims. Additionally there are concerns as to how useful residential location data is, for it gives no insight into potential points of contact with the disease, the places of transmission and contraction. It provides no material on the interactions of the city's residents, their activity and action spaces within the city or the types, duration and length of movement through the city that people had. The data set is rather small, only 127 cases total, and has limitations that render making simulations of the passage of the affliction and detailed examinations of the disease's diffusion difficult. Furthermore, there is only partial information regarding the occupations, assessment and income taxation data.

The newspapers have not only provided the temporal aspect of the disease they also give a spatial facet of the mortality related to the epidemic. However,

there are problems with this spatial aspect. This information, from the newspapers and the directories, almost invariably relates to the residential location of the deceased. The residence is unlikely to have been the only source of infection. It is likely to have been important as families tended to be larger and thus transmission within the family would be important, in addition to the fact that many people, particularly women, did not work and would have spent much time in the dwelling. The gathering points, the workplace, the stores, the schools, the street cars, the churches, etc. could also be the points of contact with the disease. Thus the pattern of mortality produced here may not reflect the pattern of morbidity. People may not have died where they contracted the disease. Thus it may not be possible to produce a definitive geographic spread, but the temporal spread is known, as is the extent and and type of socio-economic areas hit. From this information it is possible to get an idea of the geographic extent and those types of people afflicted. There are questions regarding the origin of the disease, contiguity of the spread and other aspects of the diffusion of an infectious respiratory disease, such as influenza, that can be asked but encounter difficulties, as Meade *et al.* (1988:241) discuss:

> It is often multinodal in character; diffuses rapidly because of its short incubation period and ease of transmission; is beset by a myriad of influences, with contagious, relocation, and hierarchical diffusion components; and is sometimes inaccurately counted or not recorded at all. In addition, individual outbreaks may

> represent the interplay of different strains, obscuring the impact of community barriers.

In this study, as could be expected, many of these obstacles are encountered.

Thus there are many problems that can beguile the historical researcher. The disparity between sources is but one. Whereas the *Telegraph* nominates one hundred and thirty victims, the *News Record* names one hundred and twenty six. Furthermore, when the obituary notices from the newspapers were examined, there is another group of people who are named as victims of the malady but do not appear in the published list. However, the different sources also act as validity checks.This problem is compounded by other sources naming victims that never appear in the newspapers. For instance, Campbell in his 1986 register of doctors in the region names two of the city's doctors as victims of the disease. (Campbell, 1986) However, their names never appear in the contemporary newspapers as fatalities of the scourge. Dr. Faulds, as a physician, would have been of prime importance in such a crisis but the papers give no indication of his fate. Could this be a further extension of the morale-boosting efforts of the newspapers noted earlier? Or is it simply a case of under-reporting? Weiler claims that the "newspapers seemed to have made a conscious effort to keep the public spirit up. Aside from citing the many good deeds of various citizens, both continually made the situation seem less glum" (Weiler, 1988:42).

Methodology

From the extant data it is possible to describe the roles of the social structure of the city, the role of manufacturing in spreading and/or combatting the disease, the politics of the time, the state of medical knowledge, the fact that Kitchener introduced quarantine against the advice of the Provincial authorities and the role of the media. Thus the pandemic shall be examined with these factors in mind. Within the constraints of the data I shall compare Kitchener's experience with that of the rest of the world. Among the questions to be considered is that of any relationship between the socio-economic status of the victims and mortality. Did those who died tend to come from any particular socio-economic class? Were certain occupations and/or workplaces more hazardous than others? Were the deceased evenly spread throughout the community or were certain groups or areas overrepresented? This questions shall be considered in addition to description of the spread of the disease through the city.

Describing and analysing epidemics

Curson and McCracken (1989:11.6) identify a number of steps for the medical geographer to take in order to adequately describe and analyse an epidemic. The first stages of this are:

Establish the existence of an epidemic: check the incidence of disease compared with some normal period - search for a surge of new cases (deaths) in some restricted time-space format . . .

a. plot the temporal distribution of cases, deaths (days, weeks, months) - examine the character of the epidemic curve.
b. plot the spatial distribution of cases/deaths - including their spatial diffusion over time.

Was there an epidemic? Was there a surge in the number of cases? Normally a small number of cases could be expected in any given year. In 1918 at least 127 fatalities occurred. Indeed, this was not an epidemic but rather part of a world-wide pandemic. The surge in deaths in Kitchener, and everywhere else around the globe, with the single exception of Tristan da Cunha remote in the south Atlantic (McGinnis, 1977:126), indicates that the disease was epidemic on a global scale. In Kitchener the associated mortality occurred in the month of October, 1918, with no deaths recorded prior to the 1st of the month and none after the 31st. The spatial facets of this pandemic are examined at the global, continental and national scale in Chapter 3. It is in Chapter 5 that consideration of the spatial aspects of the disease in Kitchener shall be presented.

Disease diffusion

Stemming from the works of Hagerstrand, Gould (1969) and Abler *et al.* (1971) diffusion, the spread or movement of ideas or phenomena over space and

through time, has been a major concern of geographers for several decades now. Diffusion implies the spread or movement out from a starting point. The diffusion of disease has been a major preoccupation of many geographers, as mentioned in the literature review (Chapter 2).

Infectious disease diffusion nearly always contains a component of contagious diffusion. Contagious, or epidemic, diffusion requires close or direct contact for transmission. The spread

> takes place in a centrifugal manner from the source location(s) outwards in a pattern which emphasizes the importance of proximity and interaction between actual adopters (acceptors, infectors, etc.) and potential adopters. (Goodall, 1987:126)

Furthermore, contagious diffusion is always an expansionary process and is strongly influenced by the "frictional effect of distance " (Abler et al., 1971:391). The 'neighbourhood effect' is very important in such diffusion, the greater likelihood of spreading to someone or somewhere nearby than to a remote location.

The pattern of diffusion is determined by the networks that encourage diffusion and the barriers that modify that movement. For infectious disease diffusion, factors including incubation period and infectivity are also of great

importance. Diffusion of disease is often considered a stochastic process with the element of chance. This has led to the development of models of diffusion that allow us to simulate the course of an epidemic. However, to produce such models it is necessary to have certain data. These include information such as origin of the disease, what vectors may exist, how fast is the transmission, how often can one infector transmit and how rapidly, what is the mechanism of spread, what threshold exists in terms of population and what is the probability of contact?

Another aspect that is important in modelling diffusion is that of barriers. Barriers play an important role in modifying or stopping the dispersal of the idea or phenomena. In this study quarantine may be considered a potential barrier to the spread of the disease, and this shall be examined.

In this study much of the information required to construct a model for the influenza pandemic of 1918 in Kitchener is simply not available. Consequently, the examination of the diffusion of the disease is limited to less sophisticated means, namely the production of a sequence of dot maps of known cases of influenza mortality over time.

Cartographic display and analysis have long been used in geographical examination of disease morbidity and mortality. Howe (1971:20-21) cites the use of dot maps to illustrate the distribution of yellow fever in New York in 1798. John Snow's map of cholera in 1854 London, demonstrating the relationship between the disease and water, is quite possibly one of the single most famous pieces of cartography. From these beginnings geographers have gone on to develop a myriad of techniques for presenting and analysing the spatial aspects of disease.

Ideally the data would avail itself to the quantitative analyses described by various researchers (Cliff *et al.* 1986, McGlashan 1983, Pyle 1979, Pyle 1980, Pyle 1986, Selby 1982, Spicer and Lawrence 1984). Possible directions for geographic analysis of such data include diffusion studies, cluster analysis, population potential, and point pattern analysis. However, the extant data does not permit such analysis. Thus, with this limitation it is necessary to adopt other approaches.

Here various scales of geographic analysis - global, continental, national and local shall be examined. Much of the information regarding the global through national levels is to come from previously published works, to put this epidemic in context, and also to examine the dynamics of influenza mortality. Following this is an examination of the spatial nature of the disease in Kitchener, utilising

the extant mortality data to portray the movement of the disease through the city. Further analyses, such as the depiction of clustering around factories are also made, as are examinations of the mortality by age groups and the role of political and social factors.

The deaths can then be mapped in a number of ways. For example, maps for each day or week could be produced indicating that period's deaths or maps displaying isomorts (isolines of deaths), as explored in Chapter 5. Much of this analysis would be visual, based upon simple comparisons of maps, whereas others lend themselves to numerical analysis. However, little use of quantitative analysis has been made as the dataset is so small to make such analysis difficult to interpret with confidence. If the data were available possible routes could include modelling or other quantitative analysis. However, as has been noted the extant data is incapable of supporting such analyses. Consequently the approach taken here includes mapping of the fatalities by place of residence and some presentation of the movement of the disease through a sequence of maps depicting the fatalities by date. Differences in spatial arrangement of deaths by age and occupation shall also be examined.

These patterns will then be compared and subjected to a social analysis of the city. The occupational status of the victims and/or their spouse or parent(s)

will be examined, as shall the socio-economic nature of the city. Did the disease display any socio-economic differentiation here in Kitchener? Further examination of the role of the manufacturing and other industry shall be made in the following chapter. Were those working in the factories overly represented in the death toll? Or were they in fact conspicuous by their absence? Was mortality clustered about the factories, was it concentrated in lower class neighbourhoods or was it evenly spread throughout the city both in terms of space and class?

Thus, following on from this brief introduction, I shall now examine the literature (Chapter 2) regarding influenza, the pandemic of 1918-19, the Canadian experience, particularly that of Kitchener, and geographical analysis, specifically of influenza. From this I shall then consider influenza itself, followed by an inspection of how the 1918-19 pandemic spread at various levels, including global, continental and national (Chapter 3). Then the stage shall be set for our detailed scrutiny of the city of Kitchener and how it experienced the dreadful events of October 1918 (Chapters 4 and 5).

Chapter 2. A review of the literature.

Physicians of the utmost fame
Were called at once, but when they came
They answered, as they took their fees
'There is no cure for this disease.'
Hilaire Belloc

For a disease that had such a massive impact globally there is an amazing paucity of material relating to the pandemic, particularly from the geographical perspective. In examining the literature, be it pertaining to influenza or the local history of Kitchener, it is striking how little mention is made of the 1918-19 pandemic. Here we have one of the three most devastating epidemics in history, and it occurred in living memory, yet what do we have to show for it? Virtually nothing. In the local histories, and in our collective cultural memory, it has slipped away leaving the merest of traces. In the influenza literature the pandemic is noted but with relatively little said on its origins, its spread or its crushing impact on the human population. However, this is not to say there is no literature regarding influenza or Kitchener. Indeed there is a most abundant literature on influenza and a great deal written on early twentieth century

Canada, but little of this material refers to the 1918 pandemic. Briefly, I shall set out to review this material in the context of this piece of research.

Influenza

There is a voluminous medical literature regarding influenza. However, references to the 1918-19 pandemic (Beveridge 1977, Galishoff 1969, Grist 1979, Kilbourne 1977, Kilbourne 1987, Marks and Beatty 1976, Monto 1987, Osborn 1977, Ravenholt and Foege 1982, Starr 1976, Stuart-Harris *et al.* 1985) and any spatial considerations of the disease (Beveridge 1977) are few and far between in the medical journals and writings. One recent work warrants being singled out for its meritorious contribution. Kilbourne's recent volume (1987), in which he undertakes the ambitious task of distilling our knowledge of influenza into one book, probably represents the state of human knowledge of the disease most adequately.

The vast majority of the medical material centres around questions of the virus itself since it was isolated in 1933. Questions regarding the virus' structure and the ever-changing nature of that structure as the virus undergoes antigenic drift and shift and the prevention, management and possible cure of the disease. There is great consideration made of isolating the virus, particularly as the virus perpetually evolves into a variety of strains. The identification of the various

strains, the mechanisms of change, recognition of the apparent periodicity of epidemics and pandemics, investigation of other animal hosts and/or reservoirs of the disease - all of these are common threads through much of the literature. Thus much of the literature has tended to have clinical application or be focused upon the molecular biology and biochemistry of the various strains of the virus that have emerged over the last few decades. It is from this literature (Beveridge 1977, Douglas 1987, Galishoff 1969, Gallagher 1969, Kaplan and Webster 1977, Kendal 1987, Kilbourne 1977, Kilbourne 1987, Marks and Beatty 1976, McDonald 1967, Monto 1987, Schild 1977, Schoenbaum 1987, Stuart-Harris *et al.* 1985, Webster and Laver 1972) that one can develop an understanding of the disease itself, how it is ceaselessly changing, the medical and epidemiological consequences of those changes and perhaps how to manage the disease, all of which were considered in the previous chapter.

However, influenza has not been the sole preserve of the medical profession and the molecular biologist. Others, including historians and geographers, have interested themselves in the malady. (Howe 1977, Meade *et al.* 1988, Patterson 1986, Pyle 1979, Pyle 1980, Pyle 1986) They help expand our knowledge from the purely medical and scientific to see how this disease has acted upon people in

the past and how it moves through populations. They also tend to provide us with the best information on the 1918-19 pandemic.

1918-19 pandemic

Within the medical literature, as was noted above, little has been written on the 1918 pandemic. Indeed this is true of virtually all literature from all fields be they medical, historical or otherwise. Why? Much that was written then has, as Beveridge notes, proven to be farcical later when the influenza virus was isolated in 1933 as they "contain a curious mixture of obscure reasoning and jargon" (Beveridge, 1977:3-4). Could it be that the profession has unconsciously ruled out that pandemic, decided to move on, to look into the virus and its ever-changing moods? Has it decided to shift its collective attention to prevention and cure of current and future influenza cases and regards examination of past epidemics, particularly those we have so little information about, as being irrelevant or a waste of time and resources? Or is this simply part of our collective amnesia? The pandemic has disappeared from our cultural memory as authors and composers found no place to record it in their songs and writings as Crosby notes (1989:314-319). After all, it was only influenza! Due to the high morbidity and relatively low mortality (as with all influenza) the whole pandemic seems to have gone practically unnoticed.

Fortunately this appears to have changed a little of late, with a number of papers and works appearing documenting, each making their individual contribution, this vast scourge of 1918-19. (Andrews 1977, Belyk and Belyk 1988, Beveridge 1977, Collier 1974, Crosby 1976, Crosby 1989, Fincher 1989, Galishoff 1969, Gallagher 1969, Grist 1979, Heagerty 1928, Howe 1977, Kaplan and Webster 1977, Kendal 1987, Kilbourne 1977, Kilbourne 1987, Lederberg 1991, MacDougall 1984, McGinnis 1976, McGinnis 1977, Marks and Beatty 1976, Meade *et al.* 1988, Monto 1987, Pettigrew 1983, Pyle 1986, Ravenholt and Foege 1982, Schild 1977, Starr 1976, Stuart-Harris *et al.* 1985) These works all have contributed to our knowledge of the pandemic by supplying information on matters including the impact of the disease on various communities, what virus is believed to have been responsible, conjecture on the origin of the disease, scant account of how it moved around the world and particularly on the experience in the United States of America.

Crosby's 1976 work, reprinted in 1989, appears to have been the touchpaper that has kindled an interest, throughout North America, in this unhappy episode in our not too distant past. And it is Crosby's tome that stands out, it is the only truly encompassing work in that the majority of pieces concentrate on the disease in one specific location (Andrews 1977, Belyk and Belyk 1988, Fincher

1989, Grist 1979, McGinnis 1977, Starr 1976) or otherwise consider only particular aspects of the 1918-19 pandemic (Grist 1979, Kaplan and Webster 1977, Kendal 1987, Kilbourne 1977, MacDougall 1984, Monto 1987, Pyle 1986, Ravenholt and Foege 1982) or are simply brief overviews. (Beveridge 1977, Galishoff 1969, Gallagher 1969, Heagerty 1928, Howe 1977, Kilbourne 1987, McGinnis 1976, Marks and Beatty 1976, Meade *et al.* 1988, Schild 1977, Stuart-Harris *et al.* 1985) Crosby, while using a number of cities, in addition to some emphasis on the American military's experience, to give some detail, is one of the few who considers the pandemic on a larger scale. Collier's earlier work (1974), while also global in scale, tends to the anecdotal and is poorly and confusingly structured. Due to its chronological basis, the narrative shifts back and forth across the globe, returning to individual's stories as time moves forward. Each tale he tells to illustrate the pandemic is peppered throughout the book, thus resulting in a rather frustrating work for the reader. Collier (1974) brings out the human element of the saga. He illustrates the universality of the epidemic as it struck people around the world.

Origin and spread of the 1918 pandemic

One aspect of the pandemic that is of particular interest from a geographical perspective is the spread of the disease around the globe. Where did it originate

and how and where did it spread? Unfortunately this is yet another area where one encounters a discouraging lack of information. Only once in this literature is a map of the world produced, and a rather prefunctory one at that (Beveridge, 1977:41). Furthermore, what little information does exist (Beveridge 1977, Crosby 1976, Crosby 1989, Kaplan and Webster 1977, Kilbourne 1987, Marks and Beatty 1976, Pyle 1986, Schild 1977, Stuart-Harris *et al.* 1985) is very patchy in that the spread of the disease is only partially known and much is supposition. Also this material is, in places, contradictory, particularly when considering the origin of the disease. These questions of origin and the global distribution of the disease were considered in some detail in the previous chapter.

United States of America

Much of the recent material published on this pandemic has concentrated on the American (United States of America) experience. (Arrington 1990, Crosby 1976, Crosby 1989, Fincher 1989, Galishoff 1969, Kaplan and Webster 1977, Kilbourne 1977, Kilbourne 1987, McPherson 1990, Meade *et al.* 1988, Noll 1989, Osborn 1976, Pyle 1986, Schild 1977, Starr 1976) While much of this material may be of interest and merit it should not be construed as being indicative of the Canadian experience or, even less so, of the experience elsewhere around the globe. The vast majority of this material has been prompted by Crosby's work,

which has a distinct American focus. This is shown in the authors' tendencies to take a similar approach, paraphrase Crosby, and then just add the experience of their particular city or region of interest (Arrington 1990, Grist 1979, McPherson 1990, Noll 1989, Starr 1976) or by simply summarising the national experience. (Fincher 1989, Galishoff 1969, Meade *et al.* 1988, Osborn 1976)

However, a number of works from other areas have utilised the American experience but have application elsewhere. Pyle's ongoing investigation of influenza diffusion (Pyle 1979, 1980, 1986) has included examination of the 1918-19 pandemic (Pyle 1986). Kilbourne (1987), in documenting the totality of current knowledge of the disease, has made special mention of the American situation, with which he has the greatest familiarity. The influenza scare of 1976, in which swine influenza virus was found to have started an outbreak at Fort Dix, New Jersey and prompted fears of another massive epidemic along the lines of the 1918-19 pandemic, also must take some credit for leading to the publication of some of this material, and the American emphasis therein. (Kaplan and Webster 1977, Kilbourne 1977, Osborn 1976, Starr 1976). Thus the American literature is of interest to us studying the disease as it spread across the globe and the North American continent and also details the disease elsewhere against which Kitchener's experience can be compared.

Canada

Unfortunately the literature on the Canadian predicament has not had the benefit of such a volume of scholarship as in the United States. Indeed it seems that a mere handful of historians have displayed any interest in the field. Whereas the American literature includes contributions from medical writers, biologists, geographers, and historians the Canadian literature is dominated by a smattering of historians, which has particular ramifications. These works tend to be either extremely localised, usually dealing with a single city without reference to the greater national, continental or global context, or are anecdotal. (Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, McGinnis 1977, Pettigrew 1983, Weiler 1983) These all add to our knowledge of what was happening in particular places but give little insight into what is happening nationally or how the disease spread. Surprisingly in none of these works does a single map appear indicating how the disease moved across the country or the city being examined, an oversight that this study attempts to rectify.

Pettigrew (1983) "produced a fast-paced and moving account of the sufferings" (MacDougall, 1984:97) of Canadians. While Pettigrew does cover all of Canada in her study she stresses how Canadians united against the scourge and she "has added to our understanding of folk medicine and provided a series

of heroic vignettes", (MacDougall, 1984:97) it is unsatisfying in that it fails to either analyse the disease as it was played out or the methods used to combat it, nor does she place it in a larger context, as MacDougall complains "we are left seeking in vain for a systematic analysis of either the disease or the methods that were used to control it." (1984:97)

Other works on the Canadian experience of the pandemic tend to be extremely localised or merely superficial (Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, McGinnis 1977, Weiler 1988), focusing on the disease as it impacted on a single city or simply providing an overview. Andrews (1977) and Belyk and Belyk (1988) both examine the Vancouver experience, with Andrews being much the more praiseworthy effort in that she presents a great deal of material from primary sources that does not appear elsewhere. Indeed Belyk and Belyk's effort contributes little new material and in comparison seems clumsy in its understanding of the disease and handling of sources. Braithwaite's 'flashback' for MacLean's magazine in 1953 is superficial and anecdotal and also provides little that has not appeared elsewhere. McGinnis' 1977 work is a look at one town, Calgary, and while interesting is of far less value than her earlier work (1976) which provides a useful, albeit brief, overview of the entire Canadian experience. Heagerty's 1928 review of the medical history of Canada also

provides another brief summary. This is particularly useful in that it is temporally closer to the incident than anything else available although it reflects the contemporary thinking of influenza's causes and distribution, some of which is now known to be erroneous. This work provides some detailed figures on incidence and mortality along with some insights on the origin and spread of the disease across Canada. The errors in Heagerty's work relate to the origin of the pandemic, claimed to be Spain, and the cause of the disease, then ascribed to Pfeiffer's bacillus. As Beveridge comments it is "interesting and salutary to read, in the light of present day knowledge, those text-books written in the 1920s. They contain a curious mixture of obscure reasoning and jargon not so very different in character from what one reads now about those aspects of influenza we still do not understand." (1977:3-4) Furthermore, Heagerty's work is not referenced, statements are made with no reference to original source, rather he simply provides a bibliography of sources.

Kitchener

As we descend in scale the volume of literature decreases appreciably. There is markedly less material on the 1918-19 pandemic than on influenza as a whole, there is less again concerning the global spread of the disease and even less on the American ordeal, and the Canadian material is very scarce. This situation

reaches its nadir when one considers the material regarding the city of Kitchener. The local material (Conrad 1987, Donohoe 1954, English and McLaughlin 1983, Hagmeier 1981, Moyer 1979, Pettigrew 1983, Rowell *et al.* 1982, Taylor 1990, Uttley 1937, Weiler 1983) contains almost nothing on the ravages of influenza in October 1918. This is amazing considering that, as we shall see further, the city was hard-hit by the disease with the death rate increasing almost sevenfold for that month. There are very few references to the suffering and its impact at all. (Hagmeier 1981, Pettigrew 1983, Weiler 1983, Taylor 1990) Hagmeier, a local physician of the time, interviewed in 1981, had the barest of recollections, and Taylor, in writing a brief history of the local Isolation Hospitals, discounts the epidemic with one short sentence: "In 1918, the buildings were used in the influenza epidemic under the supervision of Mrs Anna Hamilton, a nurse." (1990:77) Pettigrew's references to Kitchener are slight and contribute little to our knowledge, they are simply illustrations of the Canadian plight.

Thus it is Weiler's recent (1988) undergraduate history thesis that has any sustained reference to the bout of influenza that rocked Kitchener earlier this century. This can be considered a useful, albeit brief and somewhat superficial, introduction to the Kitchener ordeal. Otherwise, once again, the pandemic and its effects are conspicuous in their absence. Nowhere is there mention of any

Illness that has ever affected as many people or seen the death rate soar in Kitchener to the extent that influenza did in October 1918, yet there is effectively no record of this event. This slipping away of all memory of this episode is noted by others throughout North America (Andrews 1977, Crosby 1976, Crosby 1989, Fincher 1989). Andrews succinctly captures the situation thus:

> Indeed, the epidemic experience as a whole was remarkably ephemeral. Those who were ill recovered in a matter of weeks or at most months. Although the financial distress and family disruption which often accompanied death continued to affect the community for some years, they tended to be forgotten outside the affected families. . . . With spring, the epidemic experience melted into the general adjustment to peace, its social effects inconspicuous among similar ones produced by the longer ordeal of war (Andrews, 1977:43-4).

Medical geography

Over the past few decades medical geography has emerged as a dynamic subdiscipline. One of the major themes of medical geography has been a concern with the spatial distribution of disease. Recent years have seen a growing volume of material published relating to medical geography and the analysis of medical matters using geographical techniques and perspectives, of which some is of great relevance in studying contagious disease. (Cliff *et al.* 1986, Goodall 1987, Howe 1977, King 1979, McGlashan 1983, Meade *et al.* 1988, Patterson 1986, Pyle 1979, Pyle 1980, Pyle 1986, Selby 1982, Spicer and Lawrence 1984) This

approach, of modern medical ecology and medical geography has developed as "a reaction against the narrow causal perspective of traditional biomedical medicine" (Curson and McCracken, 1989:2.9) regards disease as the result of interactions between three sets of determinants, these being 'agent', 'host' and 'environmental' factors. This study follows in the tradition of much of this research in examining the spread of infectious disease through a community. However, due to the limitations of the data this study does not employ many of the techniques pioneered in the literature.

Influenza has attracted much interest of late. A number of medical geographers have turned their attention to the spatial dynamics of the disease, both now and in the past, (Cliff *et al.* 1986, Patterson 1986, Pyle 1979, Pyle 1980, Pyle 1986, Selby 1982, Spicer and Lawrence 1984) with Pyle being by far the most prolific, the most visible author. Much of this study has concerned itself with the diffusion of the disease and attempting to come to some scientific, mathematical, understanding of the dynamics of the contagion. In this study we shall acknowledge these types of analysis, however, as these tend to be more oriented to studies of modern epidemic and interepidemic influenza they are of relatively little direct use. This is largely due to their concentraction upon prediction of the movement of influenza through large populations including urban centres or

regions, not single small or medium-sized communities such as Kitchener was in 1918. Furthermore, those studies that have focussed upon smaller communities tend to revolve around modelling the disease (Damms *et al.* 1976, Elveback *et al.* 1976, Fortmann 1976). This studies tend to be mathematical models that deal with fictious data in an effort to predict disease movement and the effect of various prevention strategies in modern communities. Such models have little application in this study, as they address different questions. The depth of analysis undertaken here shall be somewhat less. This is largely due to the constraints of data. The data set is rather small, only 127 cases, and has limitations that render making simulations of the passage of the affliction and detailed examinations of the disease's diffusion difficult.

Influenza is by no means the only acute infectious disease to which geographers have turned their attention. In their examination of infectious disease widespread use of mapping and modelling of the diffusion of disease has been undertaken (Adesina 1984a, 1984b, Brownlea 1972, Cliff and Haggett 1983, Cliff *et al.* 1981, Haggett 1976, Hunter 1966, Hunter and Young 1971, Kwofie 1976, McGlashan 1977, Pyle 1969, Pyle 1986, Ray 1976, Sigsworth 1980, Stock 1976). While the diffusion of infectious disease has been a widespread theme for medical geographers it is interesting to note that much of this work

has concentrated on the regional or national scale, whereas here the focus is on the diffusion of influenza at the local level. Another common element of this research has been the use of mortality data. The majority of the work has been conducted utilising mortality data in order to map or model the diffusion of various diseases. The difficulty in obtaining reliable and sufficiently detailed morbidity data is a common one. This is especially true when considering historic episodes of disease outbreak and diffusion, as this study does. Indeed it could be said that the tendency for historic studies of disease diffusion to be at larger scales is largely determined by the nature of the extant data. Often the data is so limited that it is not posssible to map the disease's movement in close detail, rather it is only possible to make broad statements based on widely dispersed data points.

Chapter 3. Influenza.

> Diseases desperate grown,
> By desperate appliances are reliev'd
> Or not at all.
>
> William Shakespeare *Hamlet* iii,9.

Influenza, or at least something very similar to it, has afflicted the human race for centuries. For aeons we have been cursed with this short-lived, highly contagious acute respiratory disease. This plague has tormented people, not in the terrifying way of other diseases with their distressingly high levels of lethality, the rate at which a disease kills, but by striking such a large proportion of the population, rendering them feverish, shaken and aching; by what Gallagher (1969:40) terms it's

> gadfly qualities. Its origins are murkier and exits more dramatic [than other communicable diseases]. And, as a guerrilla enemy camouflages himself, influenza changes its form and appears as a baffling new disease.

Further there is the disease's propensity to infiltrate anywhere and being endemic to everywhere yet having no historic refuge or endemic focus. Gallagher cites Zhdanov as claiming that ". . . influenza is perhaps the only truly

global infection, knowing no state boundaries . . . influenza exists at the present moment as an infection of humanity as a whole." (Gallagher, 1969:40). Additionally the limited effectiveness of mass vaccination and the questionable efficacy of modern drugs enhance the disease's capability to persecute all of humankind.

Another factor that complicates our ongoing battle with influenza is that it is not a single disease. Rather there are three main types of influenza viruses: A, B and C, "alphabetically named in chronological order of their isolation and definition" (Kilbourne, 1987:26). Within these types there are literally dozens of strains known. For the purposes of this study it can be understood that we are primarily concerned with the various manifestations of the influenza A virus, for as Beveridge notes "Type A is by far the most important and most interesting and so far as we know is the only one that causes serious pandemics and the only one that occurs naturally in animals." (1977:9) Type B is thought to be a human-only virus and tends to cause illness mainly in children of school age. (Beveridge 1977, Pyle 1986, Stuart-Harris *et al.* 1985) The third type, type C, was also considered to be a human-only virus but has since been isolated from pigs in China (Stuart-Harris *et al.* 1985). However, type C is relatively uncommon and "are apparently not the causes of epidemics." (Pyle 1986) Within type A

influenza many subtypes have been identified. These have included various human, equine, swine and avian subtypes of the disease, identified by their varying antigen structures.

It should be noted that here the orthodox view of influenza is taken. Somewhat more unorthodox views, such as Hope-Simpson's latent virus hypothesis (1992) or Hoyle and Wickramasinghe's (1979) argument "that influenza viruses are not transmitted between humans or animals but descend from a galactic 'life cloud' . . ." (Cliff *et al.*, 1986:18) are not examined here.

A brief history of influenza

Humankind has been pestered by influenza for centuries, as was noted above, as epidemics have ebbed and flowed over the populations of the world. According to Kaplan and Webster "one such epidemic was recorded by Hippocrates, the father of medicine [and according to some the father of medical geography (Meade *et al.* 1988, Curson and McCracken 1989)], in 412 B.C." (Kaplan and Webster, 1977:88) This epidemic was also referred to by Livy. (Beveridge, 1977:25) The next outbreak that some contend can be "reasonably identified as being influenza alone was that of 'a certain evil and unheard of cough' in 1173" (Gallagher, 1969:42) that sickened thousands of English, Germans and Italians.

The term influenza came about in 13th Century (Gallagher, 1969:39) or 15th Century (Kaplan and Webster, 1977:88) Italy when the cause of the illness was attributed to the influence of the stars. Influenza being Italian for influence. By the 18th Century this name for the affliction had been widened to also include" *un influenza di freddo*, or an influence of cold wind," (Gallagher, 1969:39) or influence of cold, particularly a marked change in temperature, as the disease tended to appear during the winter.

1510 saw the rise of a major epidemic of influenza, one that many writers are agreed upon as being an influenza epidemic of the global proportions. (Beveridge 1977, Gallagher 1969) After rising in Africa this epidemic roared across Europe and "attacked at once and raged all over Europe not missing a family and scarce a person." (Beveridge, 1977:26). During the following century more epidemics raged, with the English 'sweating sickness' now considered to be influenza (Gallagher, 1969:43) and resulting in vast mortality as "the sweat felled millions." (Gallagher, 1969:43) Yet another epidemic occurred in 1580. (Beveridge, 1977.26, Gallagher, 1969:43) This is one of the first well-documented epidemics and is believed to have originated in Asia from where it swept through Africa and Europe. Beveridge regards this as "a definite pandemic . . . possibly the first global dissemination of the disease." (Beveridge, 1977:26) Since

this time there exists a record of a number of epidemics that have wrought havoc among the world's populations.

The English adopted the label influenza in the 18th Century, while the French assumed the appellation *la grippe*. Gallagher (1969.39) attributes the English adoption of the name to Dr. John Huxham of Plymouth, and thus for the English-speaking world this pestiferous condition has been known ever since as influenza. Prior to this the English had called "influenza the 'gentle correction,' 'the grip,' and the 'jolly rant'." (Gallagher, 1969:41) Another tag the English attached to the disease was "the newe acquayntance" (Lord Randolph, cited by Beveridge, 1977:25 and Kaplan and Webster, 1977:88) in the "misbelief that it was a new disease each time it reappeared." (Gallagher, 1969:41)

Further epidemics of influenza are known to have occurred in 1645, 1743, 1782, 1830, 1837, 1847, 1889, 1891, 1892 and 1893. (Gallagher, 1969:44-6). Each of these brought the terrifying morbidity, and mortality, of large scale influenza outbreaks. Gallagher records that in Rome in 1743 "more than 80 000 collapsed. Five hundred died in a single day." (1969:44) The 1782 incidence was pandemic, being felt in Europe, China and America. Influenza epidemics since 1700 have attracted a number of investigators (Beveridge 1977, Patterson 1986) who, in addition to those epidemics recognised by Gallagher, contend that epidemic or

pandemic situations also existed in 1729-30, 1732-33, 1761-62, 1767, 1775-76, 1788-89, 1800-02, 1850-51, 1857-58, and 1873-75. (Beveridge, 1977:27-30, Patterson, 1986) Thus influenza has had a long and painful association with humanity. This relationship looks set to continue for now and well into the future. But it was to be 1918-19 that was to see the greatest pandemic of influenza ever to stalk the planet.

What is influenza?

What exactly is influenza? Sure we all personally know - virtually every one of us has been laid low by the disease at least once. But what is the clinical description of the disease? What is it? How long is the incubation period? A great deal of the medical literature has devoted itself to answering some of these questions. Much of this material (Cate 1987, Fedson 1987, Kilbourne 1987, Mostow 1987, Ruben 1987, Stuart-Harris *et al.* 1985) is intensely technical, and for our purposes a summary of the salient points is more useful. In providing such a summary Schild (1977:350-1) succinctly answers many of these questions thus:

> In uncomplicated influenza the commencement of symptoms occurs some 2-4 days after infection. The first symptoms are headache, shivering and a dry cough accompanied by a sudden onset of fever. Malaise and aching of the limb muscles and back may occur especially in the adult patient. There may be a nasal irritation or discharge and loss of sleep and dizziness may occur. In some patients the symptoms may subside rapidly after the first 24 hours of illness. In others, the disease takes a more

> prolonged course, the temperature remaining high for 2-5 days, and the patient may have residual weakness and a cough for some days. In the absence of complications, the patient is usually sufficiently recovered to return to work within 7-10 days from the onset of illness. In a proportion of cases, persistent weakness or mental depression following the illness may require a longer convalescence. Among the clinical features of influenza the fever, 38-40°C (100-104°F) is the one which most characteristically distinguishes the disease from the common cold (an infection caused by the rhinoviruses, a group of agents unrelated to the influenza virus). (Schild, 1977:350-1)

The influenza of 1918-1919 exhibited all of these characteristics, but was far more severe than has ever been encountered before or since. (Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, Collier 1974, Crosby 1976, Crosby 1989, Fincher 1989, Grist 1979, Heagerty 1928, Kilbourne 1987, McGinnis 1976, McGinnis 1977, Pettigrew 1983, Starr 1976)

Gallagher (1969:52-3) also provides a useful profile of this acute respiratory infection with its abrupt onset, high fever, sudden chills, muscular pain, dry cough and prostration noted along with the less frequent symptoms of diarrhoea, gastrointestinal pain, head cold, sore throat, nose bleeds and blood pressure drops. Further Gallagher records that "[I]nfluenza is a self limiting ailment; without treatment it runs definite course and ends within limited time.

Recovery time is two to seven days." (1969:53) Considering the limited effects of vaccination and drugs this is probably to be considered a rather positive factor.

Influenza is universal. All ages and both sexes are susceptible to influenza. During times of epidemic between fifteen and forty percent of the non-immunised are stricken (Gallagher, 1969:53) and this figure can climb markedly under pandemic conditions (Kilbourne, 1987), as was the case in 1918. It has been noted that children and young adults appear to be most vulnerable (Beveridge 1977, Cate 1987, Gallagher 1969, Kilbourne 1987, Schild 1977). This is considered to be attributable, "in part, to the increased immunity in older individuals that results from prior infections with related viruses." (Cate, 1987.16) Other factors for higher rates of occurrence in children and young adults "may be the important role that school children play in disseminating viruses in the community." (Cate, 1987:16) This fact is of interest in examining the 1918-19 pandemic for in many communities, including Kitchener, the question of keeping the schools open was a major controversy. (Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, Crosby 1976, Crosby 1989, McGinnis 1977) In a number of communities the schools were kept open at least for a period. Vancouver, for example, initially kept the schools open as the city's medical health officer, Dr. F. T. Underhill, believed that closing the schools

"would be positively harmful to the health of children who, with schools closed, would be removed from the close surveillance of teachers and school medical staff on guard for influenza symptoms, and would instead be free to roam the streets, exposing themselves to various sources of infection and neglecting early signs of the disease." (Andrews, 1977:30)

Not only are children the most likely to be attacked by the disease but they are also rather more likely to succumb. Children, the elderly and "patients already suffering cardiac, renal, pulmonary, and metabolic diseases" (Gallagher, 1969:53) are the major groups in terms of lethality of the disease. This pattern of mortality is reflected in the hospitalisation rates as Cate recognises, that while the highest incidence is in children and young adults the rates of hospitalisation for "severe or complicated influenza are lowest, around six per 10,000, in persons five to 24 years old." (Cate, 1987:16) The highest rates are in the extreme age groups, the youngest and the eldest. Cate (1987:16) cites rates of "above 50 per 10, 000 regardless of what influenza virus type or subtype" in infants and people over 65 having "similarly high hospitalisation rates" from various studies. Cate reiterates that "[A]ge-related deaths due to pneumonia and influenza during epidemics tend to follow a pattern similar to, but some 10- to 20- fold lower than, that for rates of hospitalization." (1987:16) This pattern, of hospitalisation and

particularly lethality, is in marked contrast to that encountered in 1918-19, as we shall see shortly.

It is worth noting that it is not a rise in lethality, a rise in the proportion of cases resulting in death, that denotes an epidemic or pandemic of influenza. Rather it is "the occurrence of many cases throughout the world within a short period that constitutes a pandemic. The case fatality rate of pandemic influenza may not differ from that of interpandemic influenza, but the sudden increase of total cases is reflected by an increase in total excess mortality." (Kilbourne, 1977:1225) This may seem somewhat peculiar; if the disease isn't more lethal then why worry? Kilbourne takes up this issue, "A pandemic virus is dangerous and should be curtailed because it infects, sickens and acutely incapacitates millions and millions of people. Even if case fatality rates are low - as they have been in recent epidemics - total excess mortality will increase substantially beyond that occasioned by any other human condition." (Kilbourne, 1977:1227)

The virus itself

So what is it that causes such suffering? What does the virus 'look' like? We are fortunate in that, as Stuart-Harris *et al.* write, "[T]he past decade has witnessed an explosion of new information on the molecular biology of the influenza virus." (1985:7) and over the recent years our knowledge of the virus

has improved markedly. (Beveridge 1977, Kaplan and Webster 1977, Kendal 1987, Kilbourne 1977, 1987, Patterson 1986, Pyle 1980, 1986, Schild 1977, Stuart-Harris *et al.* 1985) Indeed, as is discussed in the preceding chapter much of the current literature is devoted to studies of the structure of the virus. So, let us now consider that structure.

This concentrated study of the virus over the past decades has revealed a spherical orthomyxovirus that is approximately 75 to 100 nanometres (nm) in diameter. Orthomyxoviruses are "lipid-containing, 'enveloped' viruses which possess the property of haemagglutination of erythrocytes, a useful characteristic in their laboratory detection and bioassay, and which contain the enzyme neuraminidase." (Schild, 1977:340) The influenza virion, or virus particle, as depicted in Figure 3-1, has a number of structural features of note.

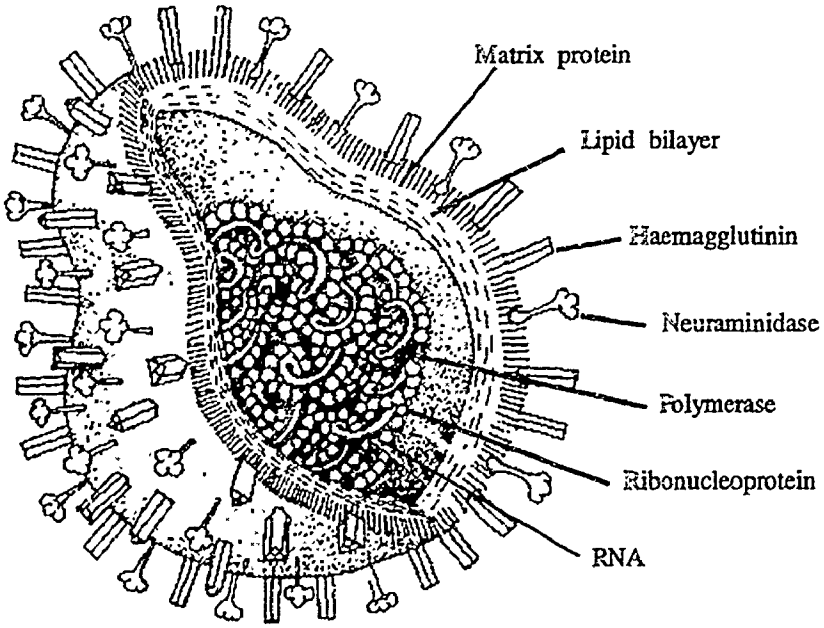

Figure 3-1. The influenza virion.
Source: Adapted from Kaplan and Webster (1977) and Pyle (1986).

The surface of the virion is covered with densely arranged radial projections, or 'spikes'. These spikes are antigenic glycoproteins with either haemagglutinin or neuraminidase activity. These morphologically distinguishable glycoprotein spikes that stud the lipid envelope are in a proportion of approximately 4:1, haemagglutinin to neuraminidase.

The haemagglutinin spike, abbreviated to H or HA, is roughly triangular in cross section and extends approximately 12 nm from the lipid membrane. This spike is responsible for binding the virus to red blood cells and host cells. In

binding to the red blood cells it causes them to agglutinate, hence the name haemagglutinin. After infection antibodies to the haemagglutinin spike are formed preventing reinfection with the same strain of influenza virus. Considerable antigenic variation has been observed in the haemagglutinin antigen. We shall return to this fact shortly, as it has serious ramifications.

The second type of spike, the neuraminidase spike, usually abbreviated to N or NA, "takes the morphological form of a mushroom but with a narrow stalk." (Stuart-Harris *et al.*, 1985:9) This spike is an enzyme that "cleaves terminal salic acid residues from glycoproteins and glycolipids" (Pyle, 1986.4) thus facilitating the spread of the virus from one cell to another in addition to the release of progeny virus from infected cells. After infection antibodies against the neuraminidase enzyme are also formed, but they are of considerably lesser importance in providing protection from infection than those produced in response to the haemagglutinin spike. The neuraminidase molecule also displays antigenic variation, however, it is less variable than the haemagglutinin molecule. Thus both the haemagglutinin and the neuraminidase spikes are highly variable proteins and antibodies to these proteins distinguish different strains of influenza virus. It is this antigenic variation that leads to new strains, subtypes and variants of the influenza A virus, as shall be discussed shortly.

These surface projections, or 'spikes', are embedded in, and through, a lipid bilayer. This lipid bilayer with its inner membrane or matrix protein stabilises the virion. Within this envelope lies the core of ribonucleoprotein, with the nucleoprotein and polymerase proteins. Indeed, there are eight ribonucleoprotein complexes. Each of these consists of "multiple copies of a single species of nucleoprotein, one molecule of single-stranded RNA, and probably one or more copies of each of three polymerase polypeptides. The ribonucleoproteins probably exist as helical structures, and each ribonucleoprotein contains one of the eight RNA segments that compose the viral genome." (Kendal, 1987:4-5)

This segmentation of the RNA, or ribonucleic acid, containing the genetic information into eight single-strand segments is somewhat unusual among animal viruses. This segmentation has major repercussions for the variability of the virus into a myriad of types, subtypes and variants. Kaplan and Webster (1977:91) succinctly note that this "segmentation of the RNA means that genetic recombination, or reassortment, can occur readily during mixed infection with different influenza A strains. The recombination of RNA segments is probably of key importance in accounting for major antigenic variations of influenza viruses" (Kaplan and Webster, 1977:91)

Variation in influenza

Recombination of the RNA and changes in the haemagglutinin and neuraminidase processes lead to changes in the nature of the virus, notably its infectivity. How does this happen and why? Patterson, in introducing his investigation of historical pandemics, concisely puts it this way:

> The influenza A genome consists of eight separate pieces of single-stranded RNA. These eight segments function as distinct genes, so genetic reassortment occurs readily when a host cell is infected with two strains of virus. Such reassortments are genetically equivalent to recombinations are . . . a major source of the virus's remarkable antigenic variation. Two of the viral genes code for surface glycoproteins, haemagglutinin (H) and neuraminidase (N), the crucial antigens against which the host develops immunological defenses in the form of circulating antibodies. (Patterson, 1986:2)

There are two kinds of antigenic variation that have been recognised. Minor antigenic change, or antigenic drift, consists of slight variations of the immunological characteristics of the surface antigens thereby producing variants. This antigenic drift is "believed to be analogous to the phenomenon known as 'genetic drift' in plants and animals." (Beveridge, 1977:71) That is, there occur minor mutations of the genetic code for these antigens, resulting in slight alterations of their properties. This is a continual process, the changes are brought about by mutations in the RNA genome.

Major antigenic changes, or antigenic shifts, such as sudden and complete change in one or both of the surface antigens, haemagglutinin and neuraminidase, result in 'new' viruses "to which the population has little or no immunity and it is these viruses that are the cause of the major pandemics of influenza." (Webster and Laver, 1972:449) The newly emerging influenza A virus possesses haemagglutinin or neuraminidase

> antigens so different from those of previously prevalent viruses that they are designated as new subtypes. The new virus almost certainly does not arise directly by mutation of the preceding virus but rather may result from genetic reassortment occurring between influenza A virus of human or nonhuman hosts, or a reappearance of a virus after a prolonged absence in humans. (Schild-Harris *et al.*, 1985:54)

So, to summarise, antigenic drift is caused by mutation in the genes coding for haemagglutinin and neuraminidase and antigenic shift is caused by genetic reassortment resulting in completely different antigens.

It these antigens that we use to identify different subtypes of influenza. Recently the World Health Organisation reorganised the nomenclature of influenza viruses. The current designations are summarised in Table 3-1, below.

Subtypes of Haemagluttinin and Neuraminidase Antigens of Influenza A Viruses.[a]

Haemagglutinin subtype	Former designation	Neuraminidase subtype	Former designation
H1	H0,H1,Hsw1	N1	N1
H2	H2	N2	N2
H3	H3,Heq2,Hav7	N3	Nav2,Nav3
H4	Hav4	N4	Nav4
H5	Hav5	N5	Nav5
H6	Hav6	N6	Nav1
H7	Heq1, Hav1	N7	Neq1
H8	Hav8	N8	Neq2
H9	Hav9	N9	Nav6
H10	Hav2		
H11	Hav3		
H12	Hav10		
H13	[b]		

a WHO memorandum (1980).
[b] Proposed as new H subtype; avian source (Hinshaw *et al.*, 1982, 1983)

Table 3-1. Subtypes of Influenza A virus.
Source: Kilbourne, 1987:27.

Obviously such changes can have a major impact as they effectively change the immunological pattern. Such changes, both drift and shift, can result in a virus to which populations have less or even no immunity. This is particularly true of antigenic shifts where the virus is changed so markedly that we recognise it as being a new subtype. It is such a major change that Kilbourne argues as

being one of the necessary preconditions for the arrival of a pandemic. He recognises three requirements for pandemics:

> (1) major antigenic mutation of the virus, (2) a generally susceptible population on the basis of immunologic inexperience with the antigen, and (3) (usually unstated) the disappearance of the immediately antecedent strain following, at the least, a ten-year period of its prevalence. The remarkably constant clinical picture of disease from epidemic to epidemic is striking. (Kilbourne, 1977:1226)

It is widely held (Beveridge 1977, Crosby 1989, Kaplan and Webster 1977, Kilbourne 1977, Kilbourne 1987, Schild 1977, Stuart-Harris *et al.* 1985) that all of these conditions were met in 1918. We believe that a new virus emerged, related to swine influenza (Beveridge 1977, Crosby 1989, Kendal 1987, Kilbourne 1987, Stuart-Harris *et al.* 1985) and it struck a population that had no immunologic experience of the new antigen(s) (Beveridge 1977, Crosby 1989, Kaplan and Webster 1977, Kendal 1987, Kilbourne 1987) and that the prior strain of the virus was replaced (Beveridge 1977, Kaplan and Webster 1977, Kilbourne 1987, Pyle 1986, Schild 1977, Stuart-Harris *et al.* 1985). Further the clinical expression of the new viral subtype whilst more severe was manifestly consistent with other subtypes of influenza, both before and since (Andrews 1977, Arrington 1990, Belyk and Belyk 1988, Braithwaite 1953, Cate 1987, Collier 1974, Fincher 1989, Galishoff 1969, Gallagher 1969, Grist 1979, Heagerty 1928, Howe 1977, Kilbourne

1977, McGinnis 1976, 1977, Marks and Beatty 1976, Noll 1989, Pettigrew 1983, Pyle 1986, Starr 1976, Stuart-Harris *et al.* 1985).

Thus, the previously prevailing virus had been out-evolved, out-competed. The new virus has an edge in survival; the lack of immunity in the host population that gives it the competitive edge and thus it supplants its predecessor.

1918-19 pandemic

"Flu claimed more lives than the four years of world war did, total war dead numbered 8,538,000." (Gallagher, 1969:48) Such a devastating event and yet it passed through history with relatively little comment until recently. (Beveridge 1977, Collier 1974, Crosby 1976, Crosby, 1989, Fincher 1989, Gallagher 1969, Kaplan and Webster 1977, Kilbourne 1977, Kilbourne 1987, Monto 1987, Pyle 1986, Schild 1977, Stuart-Harris *et al.* 1985)

'Spanish' influenza - why the name?

When this pandemic struck the disease was named 'Spanish influenza'. Apparently the epidemic "was said to have originated in Spain, hence the term 'Spanish Influenza'." (Heagerty, 1928:215) However, all but nobody believes the disease originated in Spain, rather it gained the name for a number of possible

reasons. Gallagher notes that "King Alfonso XIII (1886-1941) of Spain contracted it and this is suggested as one possible reason for the Spanish label." (Gallagher, 1969.47) However, of possible greater importance was that outbreaks were reported there with no censorship, as opposed to the countries at war, whereas in other countries, especially among the troops, it was unreported. (Marks and Beatty, 1976:271) Furthermore, Spain's neutrality made them unpopular. McGinnis is among those, in the majority, who suggest these factors as being important: "Spanish influenza did not come from Spain. That country likely acquired the blame because, not involved in the war and having no press censorship, its epidemic in the spring of 1918 was the only earlier one widely publicized. " (McGinnis, 1977:121-2) Also it has long been a common reaction in time of disease to externalise the blame onto others, in this case Spain.

How the pandemic manifested itself

The 1918 pandemic is now considered (Kilbourne 1987, Monto 1987) to have arisen as the consequence of "the appearance of a new type A influenza variant, which seroepidemiologic studies have identified as related to swine influenza." (Monto, 1987:21) Thus, it is considered to be similar to the influenza Schild describes, as Kilbourne notes that "[E]ven in the uniquely virulent epidemic of 1918, a typical case of illness resembled the 3-day fever characteristic of more

recent epidemics" (1987:5), with the enhanced virulence and lethality that a new strain has, attacking an unprepared, defenceless, population. However, the influenza of 1918 did have some notable clinical differences, "In addition to some of the classic symptoms of the disease - fever, headaches, nausea, muscle pains, and respiratory problems - many victims expectorated quantities of sputum and turned purple or blue." (Pyle, 1986:41) Some argue a case for the 1918 pandemic having been made so severe by the combination of the virus and an additional pathogen (Kilbourne 1977, Kilbourne 1987, Pyle 1986, Stuart-Harris *et al.* 1985), as influenza "epidemics are notoriously associated with an increased incidence of bacterial pneumonia . . . bacteria were major contributors to influenza complications and mortality" (Kilbourne, 1987:174). Beveridge describes how the disease manifested itself in 1918-1919:

> It is of interest to note that in the 1918-19 pandemic the illness in the great majority of cases presented the characteristic picture of influenza. Even in the severe autumn wave 80 per cent of patients suffered only the usual 3 - 5 day illness. However, often the sudden onset was more striking than usual . . . It was commonly found that about 20 per cent of cases developed pneumonia and a considerable portion of these - up to half - ended fatally. Frequently the pneumonia came on suddenly and some patients quickly developed a heliotrope colouration of the lips and face. Most of these patients did not feel especially ill and many remained cheerful but the doctors soon learnt that the heliotrope cyanosis nearly always meant death within 24 to 48 hours. This type of pneumonia, due to massive invasion of the lungs by the virus, has been

> seen occasionally during more recent epidemics. (Beveridge, 1977:15)

This vivid cyanosis, almost invariably preceding death, impressed itself upon many of those treating the ill. (Braithwaite 1953, Crosby 1976, Crosby 1989, Fincher 1989, Grist 1979, McGinnis 1976, McGinnis 1977, Pyle 1986, Starr 1976, Stuart-Harris *et al.* 1985) As Stuart-Harris *et al.* account the occurrence of this heliotrope cyanosis "or a dusky facial flush in these young persons was a terrible prognostic sign and many patients with influenza died without the picture of full pneumonic consolidation but with haemorrhagic oedematous lungs." (Stuart-Harris *et al.*, 1985:119-120)

But why did the 1918-19 pandemic take place? What changes occurred to cause influenza to become so virulent, so lethal? Schild records that the

> enormous impact of the pandemics which occurred in 1918-1920 is now legendary. It seems likely that the onset of the outbreaks in 1918 coincided with an antigenic "shift" in the influenza A virus and that this subtype virus persisted from 1918 until 1956, undergoing only antigenic drift during this period. There is indirect circumstantial evidence based on studies of the sera of persons alive before 1918, that the 1918 pandemic virus is closely related to influenza A viruses of swine . . . (Schild, 1969:344).

Furthermore, he adds that

> [I]t was found that persons who, in 1935, were old enough to have experience of infection in the 1918 pandemic possessed antibody to the swine virus; younger

> persons did not. This finding has been confirmed in recent studies . . ." (Schild, 1969:349) and ". . . there is circumstantial information suggesting that this pandemic was caused by the classical swine influenza virus (Hsw1N1) or a virus antigenically close to it. First, the initial appearance of swine influenza virus in pigs in the U.S.A. coincided with the onset of human pandemics. Second, antibody to the swine virus is present in a high proportion of individuals who were alive in 1918. (Schild, 1969:367)

Kilbourne (1977:1226) also suggests that the 1918-19 pandemic arose from a major antigenic change, probably from a form of the virus closely related to swine influenza. This virus, designated Hsw1N1, was not isolated until the 1930s, thus there is some doubt as to the real nature of the virus that prevailed in 1918-19. Attempts to isolate the virus from Inuit exhumed from the Alaskan permafrost in 1951 failed. (Beveridge, 1977:79, Collier, 1974:306, Crosby, 1989:305-6) It had been hoped that as the virus survives for long periods at sub-zero temperatures it could be possible to find the virus in the lungs of victims who died during the pandemic and had been buried in the permafrost. There are, apparently, possibilities that such work could now be repeated with a greater chance of success. But, as Beveridge warns the "remote possibility of reviving the 1918 virus must be regarded with apprehension and the strictest precautions would need to be taken to avoid any possibility of this terrible pathogen getting loose again." (1977:79)

As has been noted above it is now considered that a new subtype of influenza A, involving swine influenza virus, emerged to devastate all of humankind. However, as Beveridge records we "do not know whether or not these waves were caused by the same virus subtype. In some communities people affected in the first or second wave turned out to be immune to attack in the subsequent wave(s), but in other communities this was not so. Possibly there were two different subtypes involved or else widely different variants of the same subtype." (Beveridge, 1977:21) This issue was recently raised again by Hope-Simpson who claims that there is evidence "that at least three and possibly four influenza viruses may have been causing influenza during the period 1917 to 1919, namely two or three influenza A viruses and influenza B virus . . . The epidemic in the spring of 1918 was not necessarily caused by the agent that produced the devastating pandemic in the autumn of that year. The so-called 'third wave' in the early months of 1919 may well have been caused by the autumn virus, but here again there can be no certainty." (Hope-Simpson, 1992.27) Furthermore, the chances of our ever knowing definitively are remote.

There remain many unanswered questions regarding this pandemic. Questions relating to the virus that caused each wave or the entire pandemic, questions relating to where it originated and how it spread across the globe,

questions regarding the morbidity and mortality of this particular outbreak. Some of these questions will now be addressed.

Morbidity and mortality of the 1918 pandemic

But questions about the virus itself are not the only questions that those studying the pandemic are left to ponder. One of the most intriguing, and important, of these concerns the victims of that ghastly second wave that broke in the northern autumn. "A unique and extraordinary feature was that about half the deaths were in the 20-40 year age group and this was the pattern throughout the world" (Beveridge, 1977:31). Influenza normally has a high attack rate in young adults with its greatest mortality restricted to the extremes of age. This was most certainly not the case in the autumn of 1918. (Andrews 1977, Belyk and Belyk 1988, Beveridge 1977, Braithwaite 1953, Crosby 1976, Crosby 1989, Hope-Simpson 1992, Kilbourne 1987, McGinnis 1977, Monto 1987, Pettigrew 1983, Stuart-Harris *et al.* 1985) In this regard this pandemic was different as Monto discusses: "The 1918 outbreak was totally dissimilar, with high fatality seen in young adults. The W-shaped curve of 1918 documents the devastating impact of this pandemic, in which mortality was not restricted to extremes of age." (Monto, 1987:21-2) In this instance the young, active, productive and apparently healthy were culled from the population in apparently unparalleled

numbers. However, there is some evidence to suggest that this situation of elevated adult mortality was seen in a the epidemics of 1781-82 (Pyle, 1986:27) and 1890 (Kilbourne, 1987:8). As we see in the following chapters the disease manifested in Kitchener displayed these exact same characteristics. Many theories have been postulated as to why the young adults were taken at such a rate, including immunity issues, bacterial infections working symbiotically with the virus, or a particularly virulent strain of the virus that effectively turned the victim's immune system against the host by triggering such high levels of inflammation to fight the invading virus that the respiratory system is overwhelmed by the body's own defences. Unfortunately none of these theories seems to shed much light on the problem (Crosby 1989, Kilbourne 1987), and further "no completely satisfactory answer to that question has yet been offered - or may ever be offered" (Crosby, 1989:221).

Pandemics are defined by the unusually high rates of morbidity globally and the 1918 incident was most certainly a pandemic. "As stated, influenza generally exhibits a high morbidity but a low mortality. The 1918-19 variety was different in that it had a very high morbidity (from 15 to 50 percent in various countries) coupled with a much higher mortality rate (about 1 percent of those who contracted flu, died). It also manifested the curious feature of killing, not the

very young and the very old - its usual victims - but healthy individuals in the prime of life." (McGinnis, 1977:127) The increased morbidity is to be expected if this was a new subtype of influenza, as is thought, as the population would have little immunologic experience of this virus and thus little immunity, rendering the population vulnerable.

Variations in morbidity and mortality

Another area of doubt has been in differential rates of morbidity and mortality. That is, did the disease attack and/or slay some groups more or less than others? Unfortunately this information tends to be hidden in the immensity of the pandemic, as Beveridge suggests: "The total mortality caused by the three waves in the U.S.A. was 0.5 per cent of the population - over 500 000 deaths. In England and Wales the official figure was 200 000 deaths and in most developed countries the mortality was of the same order. In a few places the mortality was much higher. In Samoa 25 per cent of people died. The Eskimoes in Alaska suffered terribly; some villages were wiped out and others lost their entire adult population. In Nome 176 out of 300 Eskimoes died." (Beveridge, 1977:31) It tends to be among small isolated, often native, groups, that we find evidence of elevated levels of morbidity and mortality, with well-known examples being natives in Alaska and Samoa. (Beveridge 1977, Crosby 1976, Crosby 1989,

Kilbourne 1987, Pettigrew 1983) However, one should bear in mind that in these relatively small isolated communities the disease itself was probably not the sole killer, as Crosby postulates:

> Almost total isolation from humanity and its common respiratory illnesses . . . would have been enough to assure that flu would attack an immunologically defenceless population, that the majority of those infected would fall sick at the same time and the individual illnesses would be quite severe, and that for at least several days few in the infected villages would be healthy enough to provide even the barest necessities of life for the helpless. High morbidity and mortality rates are then to be expected. (Crosby, 1989:231-2)

A significant proportion of these fatalities can then be attributed to starvation, dehydration, and, where appropriate, hypothermia.

Native peoples suffered terribly, not just those isolated in far away Alaska, the high Arctic or small atolls across the globe; "American Indians suffered hideously in the pandemic . . . 24 percent of reservation Indians caught flu . . . and the case mortality rate was 9 percent, about four times as high as that in the nation's big cities. Two percent of all American aborigines died in the great pandemic." (Crosby, 1989:228)

We know that, as Beveridge does, "In this pandemic, as in other influenza pandemics, people of all socioeconomic classes from kings to beggars suffered to

much the same extent. However, the incidence sometimes varied considerably in different communities." (Beveridge, 1977:32) We also know that in the United States immigrants tended to exhibit a higher death rate than those in the country;;; this was particularly so among those born in Canada, Austria-Hungary, Poland and Russia. Italian-Americans were also found to have suffered amongst the highest mortality rates. (Crosby, 1976) But can we draw any conclusions from this? Crosby doubts so; "What can we learn from this? Probably nothing more than that some groups could afford more spacious quarters than others, and that the most recently arrived groups had a higher proportion of people of the ages most liable to pneumonic complications than groups which had arrived early. Perhaps differences in the customs of the different groups were the cause of the different death rates." (Crosby, 1989.227-8)

However, there are also some surprising elements when one examines certain populations. For example, the Afro-American population of the United States showed unusually better health than their white counterparts, as Crosby records "[B]lack Americans, locked in a caste of poverty, invariably have had a much higher death rate from respiratory disease than whites - except for the period of the pandemic of Spanish influenza." (Crosby, 1989.228) Nevertheless the bulk of evidence and opinion suggests that ethnicity itself has no direct or

causal effect on morbidity or mortality. Influenza in 1918 did appear to slay certain people more readily; "Certain groups - gasworks employees and Cornish tin miners - were only lightly touched by the epidemic; others - coal miners and pregnant women - suffered high mortality rates." (Andrews, 1977:24-5) However, Crosby provides an explanation for this intriguing state of affairs:

> There are correlations between pregnancy and death by flu, and between working as a coal miner and death by flu. But these correlations don't lead us very far. A pregnant woman has one set of lungs to handle the affairs of two bodies, and a coal miner often has something less than a fully efficient set of lungs to handle the affairs of one often overworked body. It is to be fully expected that a greater proportion of pregnant women and coal miners would die of Spanish influenza, heart disease, or anything else that might put an extra strain on the human body. (Crosby, 1989:227)

Despite all of this the question of socio-economic relations to mortality remains.

> Sometimes there was a discernable correlation between flu, pneumonic complications, and crowded living conditions - breath-borne viruses obviously more easily transmitted in cramped quarters, and the quarters of the poor are more often cramped than those of the rich - but by and large the rich died as readily as the poor. (Crosby, 1989:228)

Furthermore, could that particularly notable feature of the 1918 pandemic the "high mortality among young adults, especially males" (Hope-Simpson, 1992:27)

have any socio-economic basis? Could the lifestyles of these men have contributed to their deaths? Could their occupations, their workplaces, their places of entertainment, their living conditions have contributed to their demise? Some of these questions will be examined in the following chapters examining the Kitchener experience. Is there any relationship between the socio-economic status of the victims and the excessive mortality seen in this pandemic? Did those who died tend to come from any particular socio-economic class? Were certain occupations and/or workplaces more hazardous than others? Were the deceased evenly spread throughout the community or were certain groups or areas overrepresented?

Origin and spread of the pandemic

The pandemic is recognised as having taken place in three waves (Beveridge 1977, Crosby 1976, Crosby 1989, Hope-Simpson 1992, Kilbourne 1987, Pyle 1986, Schild 1969), starting in the northern spring of 1918. This relatively mild wave attracted minimal attention. The searing wave, the second wave, burnt its way around the globe in the northern autumn and was followed by another less severe wave early in 1919. The three waves of pandemic influenza circumnavigated the globe in a little less than a year. Beveridge concisely describes the passage of the pandemic:

> The first wave in the spring of 1918 was regarded as mild and the mortality was not unusually high. The attack rate varied with age as follows: 0-35 years, 30-40 per cent; 50 year olds, 20 per cent; 70 year olds, 10 per cent. As usual with influenza, most of the mortality was in old people, but there were also an appreciable number of deaths in the 20-40 age group. The second wave came in the autumn of 1918 and it was the most spectacular outbreak of any disease for hundreds of years. A unique and extraordinary feature was that about half the deaths were in the 20-40 year age group and this was the pattern throughout the world. The third wave early in 1919 was rather less severe but the age distribution of the deaths was similar. (Beveridge, 1977:31)

The origin of the pandemic has attracted some interest with often contradictory findings. Even after discounting the maligned Spanish we are left with a number of conflicting theories as to where the disease arose. Some postulate an Asian origin, others Russian, yet others European, some have even suggested African roots but increasing credence is given to American origins. Some of these concepts of origin and distribution are displayed in Figure 3-2.

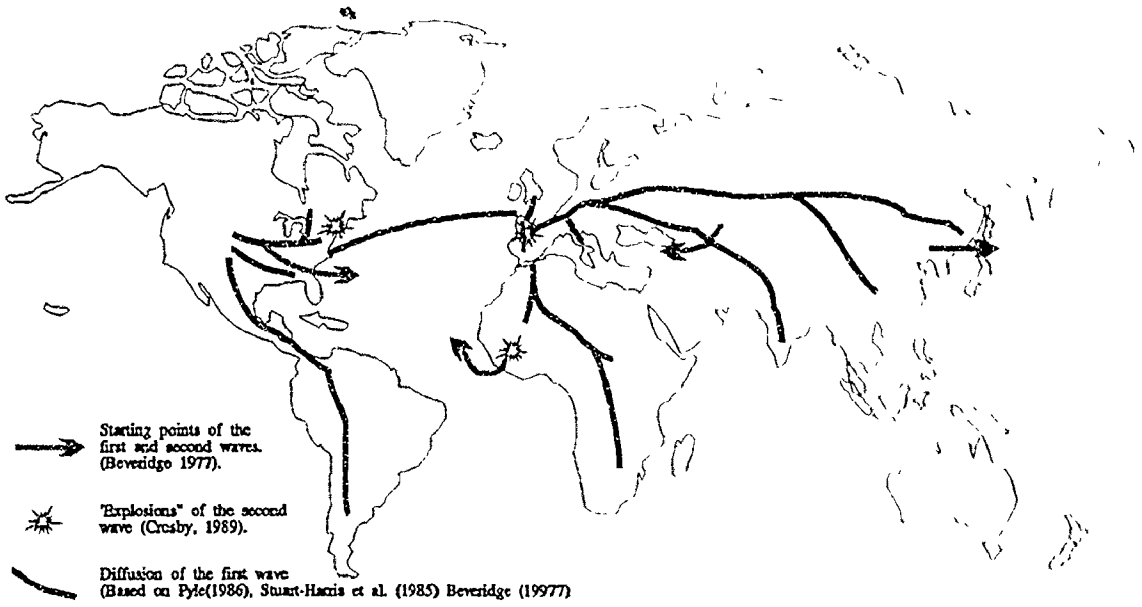

Figure 3-2. Possible origins and spread of the 1918 pandemic.
Sources: Beveridge 1977, Crosby 1989, Pyle 1986, Stuart-Harris *et al.* 1985

Asian origins?

A number of authors have suggested that Asia, particularly China, may have been the ultimate source of the pandemic subtype of the influenza A virus. (Beveridge 1977, McGinnis 1976, Marks and Beatty 1976, Pettigrew 1983, Schild 1969) This attribution of an Asian origin ranges from possibly being the source of the milder spring wave (Beveridge, 1977) and remarking that an "influenza epidemic occurred also in Japan and China in the spring of 1918." (Marks and

Beatty, 1976:271) to such sweeping statements as "it seems probable that the infection spread from Asia to Europe" (Schild, 1969:366).

Some of the support for this view comes from a particular view of the history of epidemics as Marks and Beatty explain:

> Throughout history epidemics had generally run from east to west. 'Traditionally Asia has been the matrix of disease, as if there were a permanent focus of infection that existed in the vastness of Mongolia from where it would erupt periodically into the rest of the world. Some doctors maintained that the influenza was introduced into Europe by Chinese labour battalions that landed on the coast of France. Some attributed it to Russian soldiers arriving from Vladivostock. Others thought it might have developed among the troops from an earlier bronchitis so prevalent in Spain in the spring that it gave the name Spanish to the autumn influenza. There was even one tenuous theory that the disease sprang into being in an isolated Georgia training camp during the winter of 1917 and that from there it migrated westward until it had circumnavigated the earth.' (Marks and Beatty, 1976:272-3 - citing Russell, 1958:219).

Indeed there is some evidence that epidemics may arise in Asia. According to Cliff *et al.* "three of the last pandemic strains of influenza have been isolated in Asia" (1986:27). Furthermore, their analysis of influenza diffusion "revealed that Southeast Asia could be regarded as a diffusion pole from which virus dispersion occurs to other areas" (1986:261). However, as Pyle notes this 'China

thesis' or what he terms 'xenogenic assumption' is "so pervasive in the epidemiological literature that it has become a form of dogma" (Pyle, 1986:3).

Contemporary thought sees a Chinese origin for the 1918 pandemic as unlikely and apparently unsupported by the available evidence. Certainly the influenza struck the region, as it did everywhere, but it is now thought unlikely that the disease originated here. We now consider some of the other possibilities.

European origins?

It is well documented that influenza was rampant among the troops in Europe, some see this as indicating that the disease arose there (Dowdling and LaPatra 1983, Marks and Beatty, 1976) among troops in France. However, this is viewed by most as being unlikely as there is evidence of the disease being present elsewhere before it was found in Europe. One theory suggests a Russian origin. Russia, a country that was in such turmoil at the time, embroiled in both a war and its own upheaval, has also been named as a possible fount from whence the disease sprang. However, this has been difficult to establish, in no small part due to the poor records that exist. Beveridge (1977.40) suggests, after having "scrutinised the records" that Russia may have been the primary focus or source for the severe wave of 1918. However, he also recognises that west Africa may also have been a source for this severe wave, as does Stuart-Harris *et al.*

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(1985:119). Crosby (1976, 1989) also notes that west Africa, particularly Sierra Leone, was one of the first regions to experience an 'explosion' of the severe second wave, as shall be discussed later.

American origins

Among the majority of those studying this pandemic however, the consensus now is that the disease probably originated in the midwest of the United States. (Belyk and Belyk 1988, Beveridge 1977, Crosby 1976, Crosby 1989, Fincher 1989, Gallagher 1969, Kaplan and Webster 1977, Pyle 1986, Stuart-Harris *et al.* 1985) One of the first known instances of influenza occurred at Fort Riley, Kansas on March 11, 1918 with other almost simultaneous cases reported at military installations and locations in the United States. (Belyk and Belyk 1988, Beveridge 1977, Crosby 1976, Crosby 1989, Fincher 1989, Gallagher 1969, Kaplan and Webster 1977, Stuart-Harris *et al.* 1985)

Pyle (1986) is one of those who debunks the Chinese origin theory and leans towards the USA. His reasonings for this include the retrospective analyses of the pandemic that suggest the American origin, the recorded incidences of influenza in early 1918 and the fact that the

> kind of influenza found in Kansas, Missouri, and other parts of the Midwest was different from the 'ordinary form' of the disease because mortality was

> characterized by a 'W'-shaped age-morbidity curve. In other words, in addition to higher influenza death rates among the very young and elderly, there were also elevated rates within the 20-to-40 year-old group. It was not until the next wave of influenza during the autumn of 1918 that much attention was paid to the nature of the spring wave and how it differed from other epidemics in the proportion of victims who were young adults. During March and April of 1918, the disease spread from the Midwest into parts of the South and many military camps in various parts of the United States. Probably, troops of the American Expeditionary Forces carried this form of influenza to Europe during the spring of 1918. As the spring epidemic waned in the United States, an even more virulent form of influenza with the same 'W'-shaped age-mortality curve surfaced in French port cities. The disease quickly spread to the western front, which seemed to serve further as an epicentre for an incredibly lethal 'second wave' of influenza that occurred in May 1918. (Pyle, 1986:40)

Thus the form of the disease seen in the Midwest appears to have displayed some of the characteristics that were to be seen with such disastrous consequences all over the globe in later months.

Stuart-Harris *et al.* summarise our accumulated knowledge of the origins thus:

> There is still doubt concerning the origin of the 1918 pandemic. It appeared in Western Europe in April among men of the US Expeditionary Force landing at French ports. Even before this in March, 1918, there were outbreaks of influenza in training camps in the United States and a spread of respiratory illness occurred throughout the Eastern States. In Europe, the disease

> spread from the military to the civil population and the peak of the epidemic in Britain took place in June. Though there are reports of influenza in China in March, 1918, Vaughan (1921) could find no evidence to support the existence of influenza in Asia before the time of its occurrence in America. The engulfment of the Western World in the War had brought together men from countries all over the world and this traffic could well have altered the progress of the pandemic from its usual pattern of spread in times of peace. (Stuart-Harris *et al.*, 1985:119)

And so, as Crosby concludes "then we must say that the new influenza appeared first in March and in the United States." (Crosby, 1989:25)

The second wave

But what of the later waves, particularly that of autumn 1918? Where did that come from? Beveridge takes up the story,

> Attempts have also been made to ascertain the primary focus of the virulent second wave, as this may have been due to a quite different virus from that causing the mild spring wave. A severe outbreak with high mortality occurred in Meshed in Persia at the beginning of August; this was said to have come from Ashkhabad, a city in south-east Russia. At this time Russia was disorganised by revolution so we have no information on how influenza was behaving in that part of the world. Also in August, severe disease occurred on ships serving the west coast of Africa. An English ship, the H.M.S. Africa, left Sierra Leone for England and before it reached home 75 per cent of the ship's complement had become ill with influenza and 7 per cent had died; some other ships were similarly affected. (Beveridge, 1977:43)

Crosby (1976, 1989) recognises three major foci for the crucial second wave. Along with Beveridge he acknowledges that Sierra Leone was the site of a dramatic surge in the disease's virulence. Indeed, Crosby claims that in August 1918 "epidemics of unprecedented virulence exploded in the same week in three port cities thousands of miles apart: Freetown, Sierra Leone; Brest, France and Boston, Massachusetts." (1989:37) From here the disease spread in search of susceptible hosts, soon to again traverse the globe, but in a far more lethal form than the previous season.

Thus we have seen that where the disease came from is uncertain, as too is its exact dispersal around the globe. Various suppositions regarding origins and dispersal are shown in Figure 3-2. There have been various attempts made to describe how and where the disease passed over the globe. (Beveridge 1977, Crosby 1976, Crosby 1989, Fincher 1989, Gallagher 1969, Howe 1977, Kaplan and Webster 1977, Kilbourne 1987, McGinnis 1976, Marks and Beatty 1976, Meade *et al.* 1988, Pettigrew 1983, Pyle 1986, Schild 1977, Stuart-Harris *et al.* 1985, Weiler 1988) The belief is that the American troops carrying the virus brought it to the camps and battlefields of Europe. War brings with it tremendous displacement and movement of people often in appalling conditions. This in conjunction with the tremendous virulence of the newly emerged virus and the ease of

transmission of influenza set the scene for a calamitous pandemic and thus the disease was quickly dispersed to all parts of the globe.

United States

One country that has seen recent concentrated study on the passage of the pandemic has been the United States. Here a number of scholars have laboured to improve our knowledge of this most devastating pandemic. (Crosby 1976, Crosby 1989, Fincher 1989, Kaplan and Webster 1977, Kilbourne 1977, Osborn 1976, Pyle 1986) It could be said that we now have a fairly accurate picture of how influenza spread across that country. One of those analysing this task has been Pyle, who has produced what is considered to be the best approximation of how the critical autumnal wave swept across the nation (Figure 3-3). From the "explosion" in Boston that Crosby has identified it is readily apparent that there was "a dramatic east-west progress, with rapid penetration into the interior and a lagged arrival in the southwest and northwest." (Meade *et al.*, 1988:240) Further, Pyle's investigations suggest that "a form of contagious radial diffusion took place . . . but hierarchical spread is also apparent as the disease seemed to skip from the very largest to next largest metropolitan centers." (Pyle, 1986,.43-9)

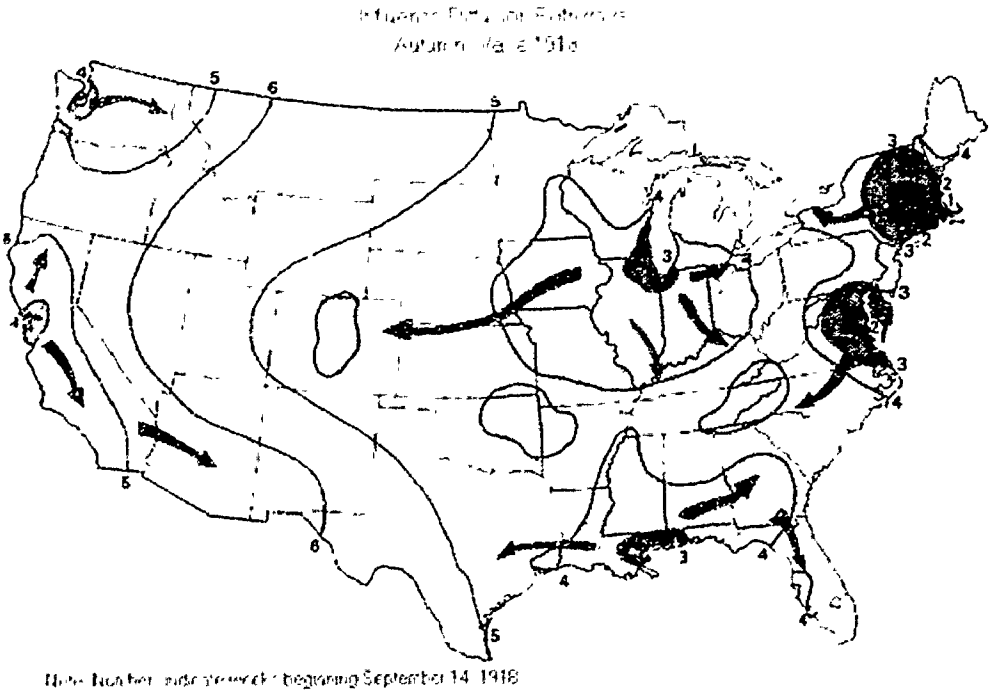


Figure 3-3. Influenza diffusion: Autumn 1918, USA.
Source: Pyle, 1986:48.

Canada

But what happened in Canada? Canada appears to have had various points through which influenza entered the country. (Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, Heagerty 1928, McGinnis 1976, McGinnis 1977, Pettigrew 1983, Weiler 1988) Overland from the USA, particularly in the east early in September, is one obvious entry. Another was via the sea. Various reports suggest influenza being brought into several Canadian cities, including Quebec

City, Montreal, Vancouver and Halifax. (Andrews 1977, Belyk and Belyk 1988, Heagerty 1928, Weiler 1988) Figure 3-4 depicts what is known of the Canadian experience.

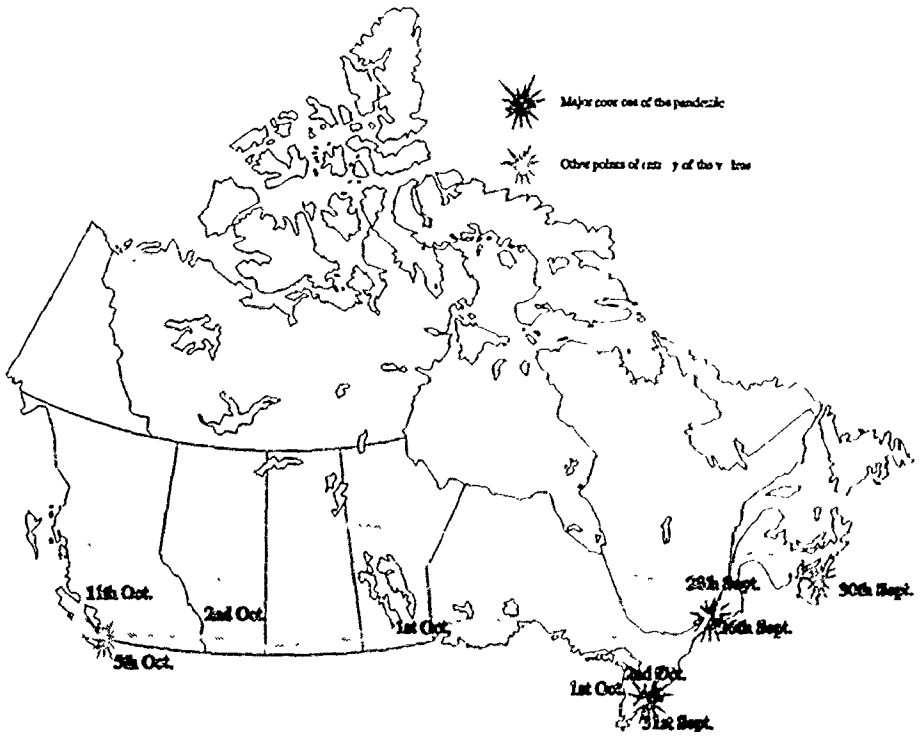


Figure 3-4. Influenza sources and diffusion: Canada, autumn 1918.
Sources: Andrews 1977, Belyk and Belyk 1988, Braithwaite 1953, Heagerty 1928, McGinnis 1976, McGinnis 1977, Pettigrew 1983.

McGinnis (1977:123), Heagerty (1928:215) and Weiler (1988:7) cite the troop ship *Araguayan* and the steamship *Somali* was being one the first ships to bear

influenza into Canadian cities. Other ships, including the *Nagoya* in Montreal and the *Med 1099* hospital ship in Halifax brought their disease-ridden human cargo into Canada also. Troops are often cited as moving the disease around the world, and this appears to be the case in Canada. One of the first outbreaks "appeared in Canada among troops stationed in the Hamilton-Toronto area" (Braithwaite, 1953) with particular attention given to some Polish soldiers in Niagara, "Spanish Influenza . . . has made its appearance in Ontario. So far, most of the cases reported in the province are in the camp of the Polish Legion at Niagara." (*Kitchener Daily Telegraph*, September 16, 1918) This Polish Infantry Camp was identified as definitely harbouring influenza sufferers very early in the Canadian experience and among these sufferers were some of the first Canadian fatalities. (McGinnis, 1977:123, Pettigrew, 1983:9)

It has been noted often that epidemics tend to "travel along lines of communication and east to west." (Pettigrew, 1983:13) In Canada this appears to have been the case as "[D]uring the months of October, November, and December the disease spread along the lines of travel and invaded even the remotest sections of the country." (Heagerty, 1928:220). The earliest outbreaks appeared in Quebec and Ontario and then the disease was dispersed east and west, as can be seen in Figure 3-4. This trend to the west was recognised

relatively early as the *Kitchener Daily Telegraph* for the 15th October, 1918 attests in proclaiming that the "Spanish 'Flu' Epidemic Is Moving Westward" on its front page. Thus after the appearance of the disease in Quebec and Ontario it dispersed across the country and the "[S]maller Ontario centres were the first to report, then those in the southern prairies. The epidemic was serious in Edmonton by 18 October and raging in the northern prairies generally by the beginning of November" (McGinnis, 1977:125)

The lines of communication, particularly the railways are considered to have been of prime importance. Indeed the Canadian Pacific Railway seems to have facilitated the spread of the disease, "The epidemic moved rapidly west along the Canadian Pacific Railway mainline and gradually spread out to the most remote areas." (Belyk and Belyk, 1988:44) Thus the disease moved westwards, "In September, . . . influenza was clearly working its way toward Vancouver along lines of travel" (Andrews, 1977:28). Moving along these lines were many troops, spreading the disease into numerous communities (Andrews 1977, Belyk and Belyk 1988, McGinnis 1976, Pettigrew 1983). Not only did Canadian Pacific Railways assist, however unwittingly, in the spread of the disease, but the disease took its toll upon the railway and its employees, as it did upon the entire populace of Canada (Pettigrew 1983).

Scrutiny of the dispersal of the disease through Canada suggests that a hierarchical element, as Pyle (1986) described in the United States and Cliff *et al.* (1986) found elsewhere, may also have been at work here. The disease struck early in the major eastern cities and from there spread through the various communities, apparently tending to strike at the larger centres earlier. This pattern was seen in many locations including the Prairies where in Calgary "after the city inhabitants decided that flu was no longer a problem many of Calgary's health personnel went to towns north of Edmonton where there were later but severe epidemics." (McGinnis, 1976:7) Hierarchical patterns were also apparently seen in British Columbia as from Vancouver "the disease spread rapidly to neighbouring municipalities, and on to other cities, towns and villages throughout the provinces." (Belyk and Belyk, 1988:45)

Chapter 4. Influenza in Kitchener - an introduction.

> When a lot of remedies are suggested for a disease,
> that means it can't be cured.
> Anton Chekov "The Cherry Orchard" II,i.

In this chapter I describe the pandemic of 1918 in Kitchener, reconstructing the passage and impact of the disease from the primary sources. This chapter follows the course of the disease, thus it is arranged chronologically with a number of important areas examined as they become apparent. The chapter concludes with consideration of the morbidity and mortality brought upon Kitchener by the disease with emphasis on the comparison between Kitchener and elsewhere around the world. In the following chapter analyses of the patterns and the nature of the disease are made.

Study area - Kitchener

Kitchener, Ontario in the County of Waterloo was already an important regional centre with a significant population by 1918. Prior to the war the 1911 population was 15 196, comprising 1.1 percent of Ontario's urban population. According to the assessment rolls of 1918 the population had reached 19 767

(City of Kitchener Assessment Rolls 1918). By 1921 this had grown to 21 763 and 1.3 percent of the province's urban population (English and McLaughlin, 1983:241) and the city was one of Ontario's major manufacturing centres, with rubber being an important industry. The city was not to escape the influenza epidemic unscathed. Indeed, there were, as Moyer (1979:59) records,

> hundreds of cases in the Kitchener area. Medical officer Dr. J.F. Honsberger later wrote that it was not uncommon to see two funeral processions whilst driving between Kitchener and Waterloo. The hospital was filled, and doctors and nurses were themselves falling victim to the often fatal illness. Dr. Honsberger notes that twenty-four hour duty was not unusual for people in the medical profession during the epidemic. But in spite of all efforts many dozens of deaths resulted in Kitchener and throughout Waterloo County.

Eileen Clarke's recollections of the time mirror this image. She remembers

> October that year, . . . seemed to me that everybody in the city was dying. They were all dying off in the flu . . . They were dying all over . . . The funerals were just one after the other (Clarke and Clarke, 1982).

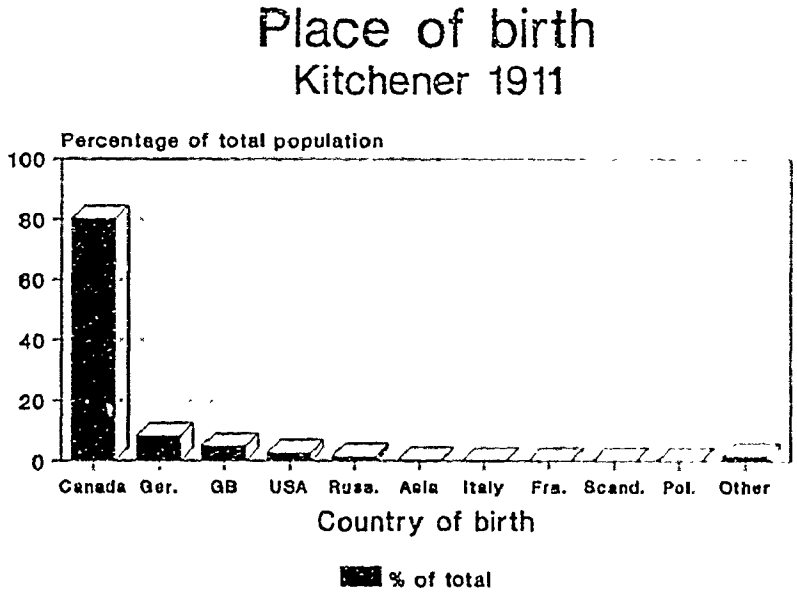
Dr. Hagmeier illustrates the privations of the medical profession when he recalls that for "three nights I didn't get to bed at all" (Hagmeier, 1981).

The rolling countryside of southwestern Ontario forms a rural heartland in which Kitchener has long been an important regional centre. The former maple and cedar swampland of the area has been transformed into a modern Canadian

city, with its ethnic melange. This transformation has come about as the settlement grew from a small Mennonite community into a contemporary North American city. Prior to 1916 the city was known as Berlin, with over two-thirds of its population of German origin (Figures 4-1 and 4-2). During the First World War the city was wracked with ethnic and political differences. Tensions arising from the re-naming of the city, the vociferous city council elections and underlying all of these, the Great War itself. English and McLaughlin suggest that not only were there ethnic divisions but also "the voting pattern in the 1917 election does reveal a strong sense of class feeling" (1983:130).

The ethnic and class differences are obviously important in the city at this time. In 1921, according to census data, 55.6% of the population were of German origins while by 1916 in the higher employment categories "those of British background make up 44 percent . . . considerably higher than the British background of the general population (28.9 per cent in the 1921 census) [Figures 4-1 and 4-2] Moreover, the British presence in 'intermediary' professions such as law, medicine, religion and education was even more pronounced (54.9 per cent)." (English and McLaughlin, 1983:131) However, to examine ethnicity and foreign-born victims in Kitchener is fraught with difficulty. Substantial components of the population of the time were born overseas and the vast

majority of the population claimed ethnic origins other than Canadian or British. However, we have no information regarding the birthplace of the victims in all but a handful of cases and to make judgements based upon surnames would be mere supposition at best. Consequently no examination as to whether influenza mortality struck ethnic groups differentially is made here.



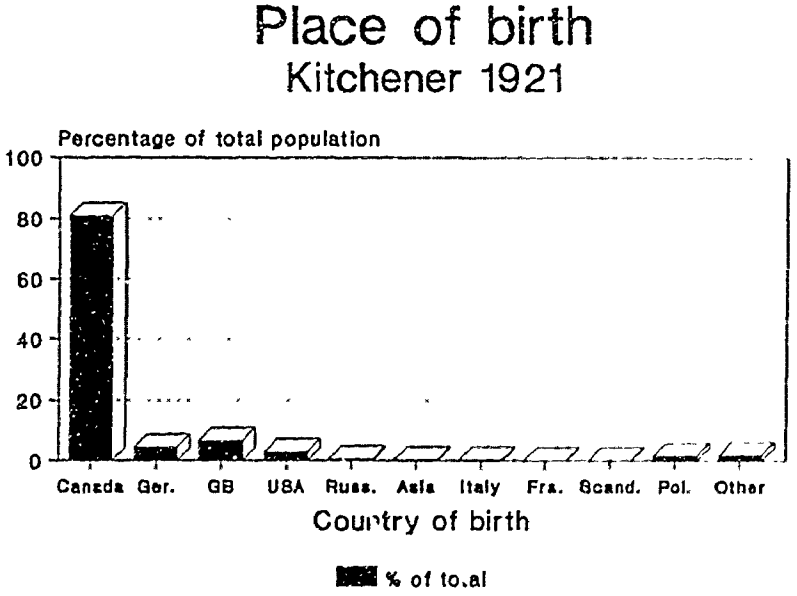
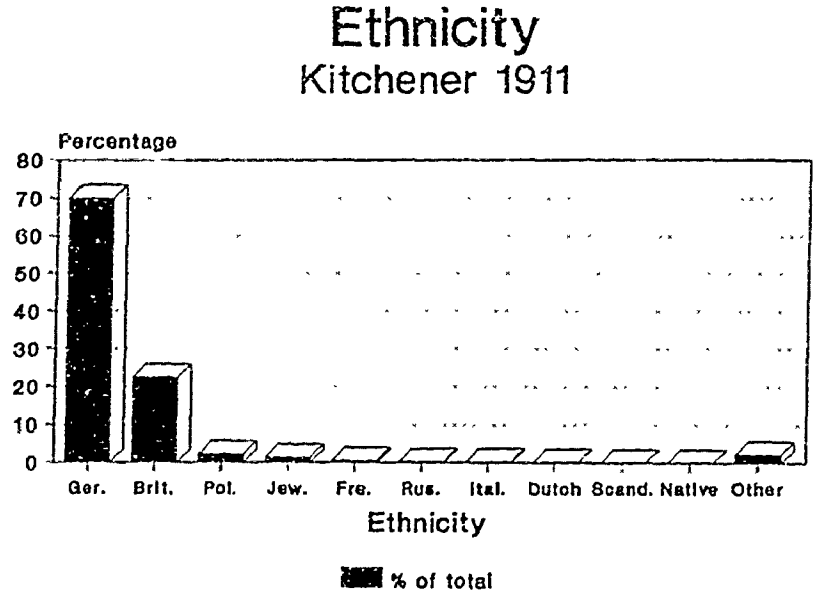


Figure 4-1. Countries of birth of Kitchener's population in 1911 and 1921.
Source: English and McLaughlin, 1983:244-5.



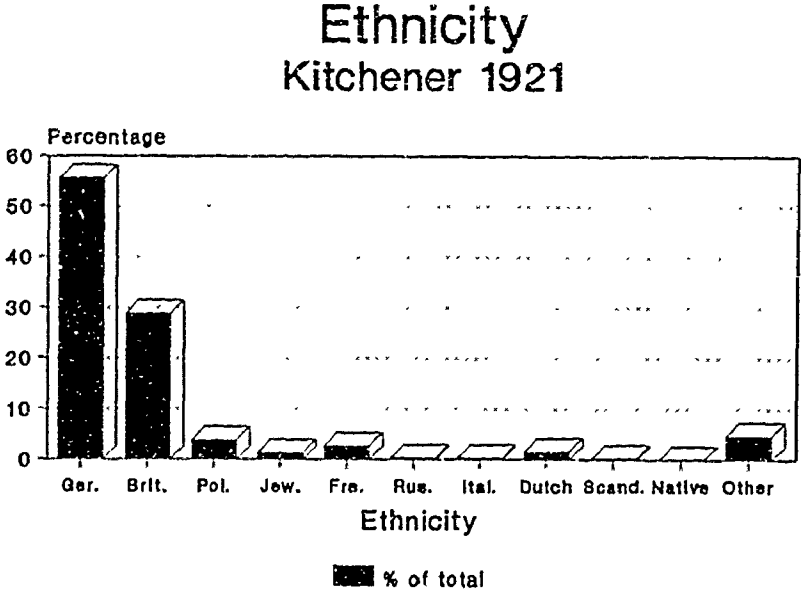


Figure 4-2. Ethnicity, 1911 and 1921.
Source: English and McLaughlin, 1983:246-7.

Influenza strikes

It is a tragic irony that the front page of the *Kitchener Daily Telegraph* (hereafter the Telegraph) of the 7th September, 1918 should trumpet that the "City is free of contagious diseases." Within a month people would be dying of what Marks and Beatty describe as the "third of the three most devastating epidemics to hit mankind." (1976:271) and within two months nearly 130 people will have died as a direct result of one of the most virulent contagious diseases known.

Indeed less than two weeks after this pronouncement of good health the *Telegraph* of the 18th September reported that "Influenza is prevalent here" as the first cases of Spanish influenza were reported in the province in a military encampment at Niagara. Another twelve days saw the first cases being reported in the city itself as the "Spanish 'flu' invades city" (*Telegraph*, September 30.1). The article suggests that the problem is minor.

However, the first death changes this. Miss Anna Hiller was to prove to be the first influenza fatality in the city as she succumbed to the disease on the 1st of October, 1918 at the home of her parents. It is from the detailed newspaper stories that one can construct an image of the impact of the disease. From the

newspaper reports a list of those known to have died in the city can be compiled (Appendix I). Then it is possible to check residential locations in the newspapers, the city directories and the assessment rolls to eliminate those who are found to have come from outside the city, thus identifying those Kitchener residents who died (Appendix II). However, detailed residential information could not be uncovered for all of these people, so they could not be mapped. Consequently this leads to a third list of victims, those for whom we have a detailed Kitchener address (Appendix III). These deaths can then be mapped in a number of ways. For example, maps for each day or week could be produced indicating that period's deaths or maps displaying isomorts (isolines of deaths), as explored in the following chapter.

The first day of October sees the front page of the *Telegraph* reporting the final days of the Great War and the spread of influenza in the city, the first death as a result of the illness and its impact on the newspaper's staff. Due to illness among the newspaper's workers the paper had to be printed by its rival city newspaper, the *Kitchener News-Record*.

Anna Hiller's death, reported by the *Telegraph* on the 1st, was followed by a lengthy obituary published in the same publication on the 2nd. Furthermore, it was accompanied by another obituary; this one recording the city's second

influenza-caused fatality, that of Mrs Emil Ruf. The Ruf's were the proprietors of the Station Hotel. Public establishments, any place where people meet, are always prime locations for the transmission and contraction of virulent contagious diseases such as influenza. Indeed the Station Hotel was the site of another death within a day as John Malineck, a bartender at the establishment, also surrendered to the contagion on the 3rd of October, 1918.

October 3, 1918 saw a number of interesting developments. The *Telegraph* carried stories recording the afore-mentioned deaths, a lead article optimistically headlined "Spanish 'flu' epidemic is on the wane" which was soon to be proven patently untrue, which also discussed the non-opening of the local isolation hospital due to a "scarcity of nurses" and a small inside page item from the Social and Personal column recording that "A number of citizens are confined to their homes with influenza, some being quite ill." This suggests that the illness crossed social barriers with ease, as can be expected with a disease as readily transmitted as influenza.

After the optimism of the front page of the 3rd the *Telegraph*'s front page of the 4th shows a return to the depressing reality of the situation. Once again the front page details the impact of the disease on the newspaper as management and writing staff had to assist in the more technical aspects of newspaper

production and again the paper had to be printed on the presses of the *Kitchener News Record*. Furthermore, another death makes front page news along with an article with the more forbidding headline that contradicts the previous day's paper, "Influenza is not abating." Indeed, the story continues to record the fact that not only are further deaths occurring but also the loss of health workers to the disease as they are incapacitated by the disease. It was at this point that the papers began printing items on how to combat the disease. The *Telegraph* also gives us the first indication of the prevalence of the disease in the various factories in the city as it reports that the

> Dominion Rubber System factories of this city have a total of 352 employees laid up with Spanish influenza. Three special nurses are working in the care of them.

The role of the factories as locations for transmission and contagion is obviously of vital importance in understanding how this scourge was visited upon the city. Time and again we return to discuss the role of the factories.

Each day of the month of October had seen a death, the 5th was to prove to be different. Today there were the first multiple deaths, two citizens fell victim on the 5th. The *Telegraph* pronounced the "Influenza situation most serious," somewhat of an understatement, on its front page. The mortality brought on by the disease was, as can be seen from Figure 4-3, in its initial stages. The peaks

were days away yet, many more were to be claimed before this illness cleared the city. This graph, Figure 4-3, is a classic epidemic curve for an infectious disease with a short incubation period such as influenza, with a sharp rise in the number of deaths which is maintained for a period before falling away almost as rapidly as it came.

Different diseases exhibit varying epidemic curves. The curves are a function of various factors, including incubation period, virulence, etc. (Lilienfeld and Lilienfeld, 1980) Fine recognises that there is "a general tendency for propagative epidemics to be 'right-skew', i.e. for the initial rise in incidence to be more rapid than the subsequent fall." (1982:47) This appears to be the case when one examines the curves for the 1918 pandemic (Figures 4-3 and 4-4). This is particularly true of the morbidity data from Montreal (Figure 4-4). However, such data must be treated with caution as it is most likely that there is drastic under-reporting of cases. These curves reinforce the idea that influenza is a fast acting disease with a relatively brief incubation period of two to four days.

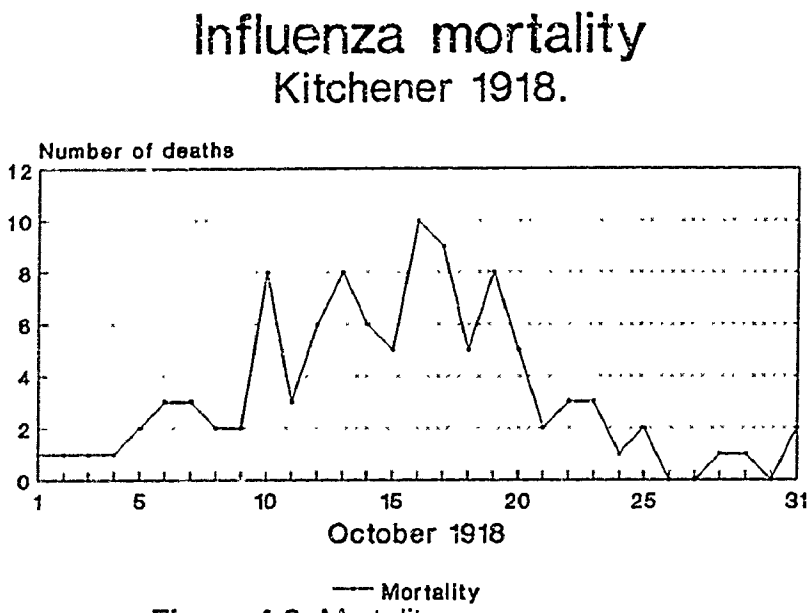


Figure 4-3. Mortality curve.

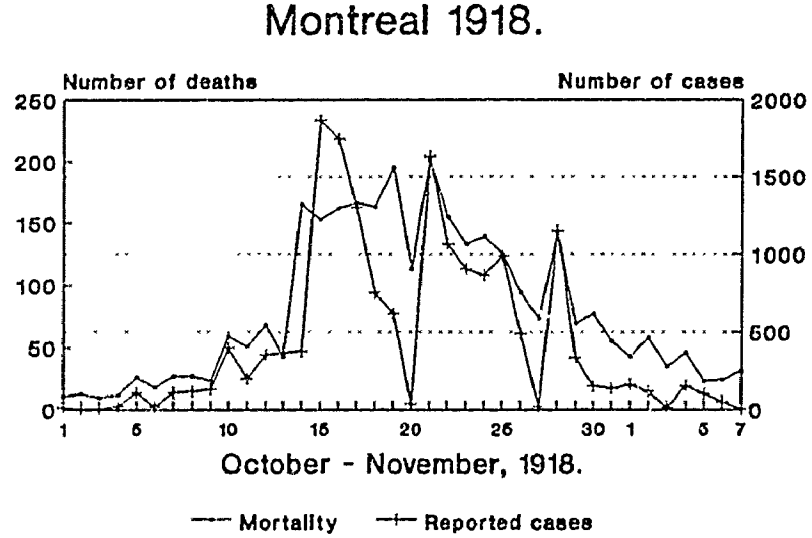


Figure 4-4. Morbidity and mortality in Montreal.

Furthermore, the shape of the epidemic curve assists in identifying the disease responsible. If the curve "rises and falls rapidly, the epidemic is likely a .

common-source epidemic from contaminated food or water, since many persons are infected in a short period of time . . . Conversely, if the number of cases over time is related to several incubation periods and the epidemic curve has a less distinct peak, the curve suggests a propagated or person-to-person epidemic" (Decker, 1988:10). Figure 4-3, the epidemic curve for Kitchener, obviously reflects the latter instance; an epidemic curve with a number of peaks related to the person-to-person propagation of the influenza with its incubation period of 2 to 4 days.

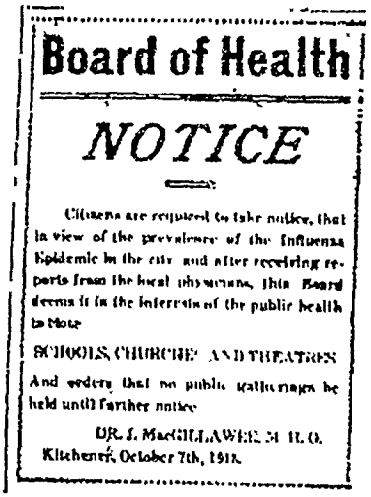
For this pandemic this pattern (Figure 4-3) was remarkably consistent, as Galishoff (1969) writing on the American experience of the pandemic noted:

> The pattern was the same in nearly every major city: one or two weeks of rapid spread followed by two or three weeks of high morbidity and mortality, whereafter the epidemic rapidly subsided. A peak period of mortality for many of the nation's urban areas, and especially New York, New Orleans, and San Francisco, occurred in the third week of October, attesting to the extraordinary contagiousness of the disease.

This is exactly the situation encountered in Kitchener (Figure 4-3) and in most other Canadian cities, including Montreal (Figure 4-4).

Quarantine imposed

The severity of the disease had become apparent to all by the morning of the 7th. The Board of Health met and pronounced that they "deem it to be in the interest of the public health to close schools, churches and theatres, and also all public gatherings, until further notice." (City of Kitchener, October 7th 1918) To this end they placed an advertisement in the city newspapers informing the populace of their decree (Figure 4-5).



Board of Health

NOTICE

Citizens are required to take notice, that in view of the prevalence of the Influenza Epidemic in the city and after receiving reports from the local physicians, this Board deems it in the interests of the public health to close

SCHOOLS, CHURCHES AND THEATRES

And orders that no public gatherings be held until further notice

DR. J. MacGILLAWEE, M. H. O.
Kitchener, October 7th, 1918.

Figure 4-5. Board of Health notice.
Source: *Kitchener Daily Telegraph*, October 7, 1918.

City closures and quarantines of various degrees were implemented in many Canadian cities. (Andrews 1977, Belyk and Belyk 1988, McGinnis 1976, McGinnis 1977, Pettigrew 1983) The efficacy of such measures has been much

questioned, and generally it is considered that quarantine and closures failed to have any significant impact upon the disease, quarantine appears to have been a very permeable barrier to continued diffusion. Obviously such a move would have a major impact on the life of the city even if little impact upon the disease. This was no longer a normal situation or a minor illness afflicting some of the population. One of the few incidents relating to the flu that the Waterloo Historical Society records was that "Our Annual General Meeting for this year had to be cancelled for the date prescribed by our By-Laws, the last Friday in October, on account of the then malignantly prevailing epidemic of the so called Spanish Influenza, and consequent prohibition of public meetings by the local Board of Health." (Waterloo Historical Society, 1918:11) This situation has been repeated all across the city, including at the Hospital where the "annual meeting was postponed owing to the influenza epidemic which passed over this community." (Kitchener-Waterloo Hospital, 1919:3)

Examining our epidemic curve, Figure 4-3, it would appear that the imposition of closures upon the city had fairly minimal impact on the mortality caused by the disease. This lack of impact is apparent when the spread of the disease is examined, as discussed in some detail in the following chapter. Here it is important to keep in mind the relatively short incubation period influenza had

and has always had of 2 to 4 days. The closures were ordered on the 7th yet peaks of mortality came about on the 10th, 13th and 16th indicating that the disease was still very much being transmitted and contracted in the workplaces and the stores after the closures were decreed. Thus quarantine proved a permeable barrier to the diffusion of influenza in Kitchener.

Other stories carried in that day's newspapers included the closure of the Kitchener-Waterloo hospital to visitors until further notice, a number of new deaths, and a major headline reading "Spanish Influenza Epidemic Reaches Crisis" (*Telegraph*, 7th October 1918:1) and the accompanying story detailing that three more deaths had been reported, "1735 cases reported by eleven out of the eighteen physicians in the city," the closures of meeting places and factories by, respectively, decree and illness and the fact that a number of the city's doctors had come down with the illness.

Kitchener's medical profession

Kitchener's doctors were overwhelmed by the disease, as were their colleagues the world over. Dr. Hagmeier was to later recall that there was little that could be done other than "Keep 'em in bed, that was all. Protect them from getting more exposure. That was about all you could do." (Hagmeier, 1981) Their

writings, their records of the period would be of immense interest if they could be consulted. We know from the newspaper quoted above, that some eighteen doctors were present. Furthermore, Campbell's (1986) inventory of the county's doctors allows us to identify many of those doctors present. Indeed examination of Campbell's work permits us to name seventeen of these men, of whom at least one perished at the time (Appendix V).

The doctor who was known to have succumbed to the disease was Dr Faulds, of whom Campbell says

> The influenza epidemic in the fall of 1918 made such constant demands upon him that when he became ill himself, he was unable to resist the disease. In this pandemic this was a common sequence of events, leading to the loss of many medical and nursing attendants. His death occurred on October 28th . . . (Campbell, 1986:84)

The other doctor Campbell identifies as being a casualty of the disease was Dr. Kirby of whom apparently little is known "but he was in Kitchener in 1918, when he was mentioned as one of the physicians ill with influenza." (Campbell, 1986:138) These men, Dr. Faulds and Dr. Kirby, as physicians would have been of prime importance in such a crisis but the papers give no indication of their fate Could this be a further extension of the morale-boosting efforts of the newspapers Weiler notes? Weiler claims that the "newspapers seemed to have

made a conscious effort to keep the public spirit up. Aside from citing the many good deeds of various citizens, both continually made the situation seem less glum." (Weiler, 1988.42) This activity was apparently quite common throughout Canada (Andrews 1977, Belyk and Belyk 1988, McGinnis 1976, 1977, Pettigrew 1983).

The disease continues

Still the *Telegraph*'s staff was wracked by illness and their October 8 edition was again plagued by staff shortages from their pressman, city and news editors right through to the delivery boys. That day's edition of the paper, when eventually printed, recorded these hardships in addition to another two deaths and the illness of a number of prominent citizens. Also reported was the "Splendid Work of Victorian Order of Nurses."

Many members of the community were volunteering their services, this situation was repeated in communities across the province (Provincial Archives of Ontario, Pettigrew 1983, Weiler 1988) and the nation (Andrews 1977, Belyk and Belyk 1988, McGinnis 1976, McGinnis 1977, Pettigrew 1983). This is borne out in the records of the Waterloo Historical Society who recorded that "During the Influenza epidemic, the Daughters of the Empire assisted the Victorian Order

of Nurses in relieving suffering." (Waterloo Historical Society, 1920:127) and by the Kitchener-Waterloo Hospital who recorded that the volunteers

> rendered splendid service, as well as a corp of graduate nurses" who helped when "the capacity of the institution was taxed to the limit during the never-to-be-forgotten period" as the staff "under the direction of the . . . Superintendent, Miss Eisele, did valiant service, and many of the nurses were afflicted with the disease as the result of their zealousness to render help as long as it was possible. (Kitchener-Waterloo Hospital, 1919:3-4)

This level of community involvement is most evident when one reads the newspaper reports throughout the month detailing what volunteers were doing, what the factories and employers did to save their workforce from the disease and is also evident in listening to the oral histories of the people (for example, Clarke and Clarke, 1982). Indeed the vital role of volunteers is well recognised even at the time of the scourge, for example, the *Telegraph* of the 26th October notes that "Over 1000 homes were cared for during the three weeks of 'flu' epidemic - Generous help of the people greatly appreciated by the Officers." (26th October 1918:1, 3)

The situation was becoming so serious that the Board of Health convened and decided that the Isolation Hospital, previously kept shut due to lack of staff, must "be used in the present epidemic of influenza, and that a resident nurse be

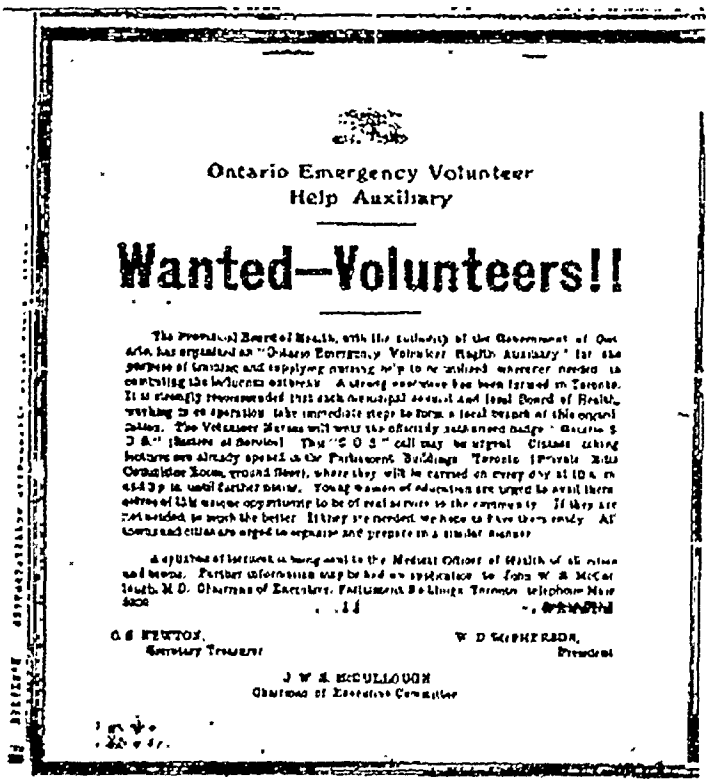
employed." (City of Kitchener, October 9th 1918) This was duly done with some rapidity, as Taylor notes, "under the supervision of Mrs Anna Hamilton, a nurse." (1990:77) This move did not come a moment too soon as on the very next day, the 10th, the first major peak of mortality was struck (Figure 4-3). Previously, deaths had been of the order of one or two per day, with an exception of five deaths on the 7th. However, on the 10th influenza claimed the lives of 10 people. The cumulative death toll climbed from 16 to 26 in a single day.

October 11 saw the Board of Health post an advertisement calling for all persons with any nursing experience to lend their assistance. The *Telegraph* ran stories appealing for "soup and broth for 'flu' victims" while also recording the growing death toll. However, also reported was a reduction in the number of Dominion Rubber System employees being cared for by the Dominion Rubber System Welfare Department and the editor headlined that story as indicating that the "Epidemic has passed crisis" - once again the paper was overly optimistic, as was the accompanying editorial.

The newspapers

Over the next few days the papers continued to list the growing toll of deaths, often accompanied by attempts at positive stories relating to the disease resulting in further headlines such as "Ladies of office staff volunteer for nursing

duty" (*Telegraph*, 12th October 1918:1) "Decrease of influenza cases are reported" and "Spanish flu epidemic is moving westward" (*Telegraph*, 15th October 1918:1). The stories of volunteers accompanied calls for such from the Victorian Order of Nurses and the Ontario Emergency Volunteer Help Auxiliary (*Telegraph*, 16th October 1918:3) (Figure 4-6).



Ontario Emergency Volunteer
Help Auxiliary

Wanted—Volunteers!!

The Provincial Board of Health, with the authority of the Government of Ontario, has organized an "Ontario Emergency Volunteer Health Auxiliary" for the purpose of obtaining and supplying nursing help to be utilized wherever needed in controlling the influenza outbreak. A strong committee has been formed in Toronto. It is strongly recommended that each municipal council and local Board of Health, working in co-operation take immediate steps to form a local branch of this organization. The Volunteer Nurses will wear the officially authorized badge "Maple Leaf" [illegible] at Service. The "S O S" call may be urged. Citizens calling between [illegible] are already opened in the Parliament Buildings Toronto (Private Bills Committee Room, ground floor), where they will be carried on every day at 10 a.m. to 4 p.m. until further notice. Young women of education are urged to avail themselves of this unique opportunity to be of real service to the community. If they are not needed to-day they may be needed to-morrow. Either way we have to save them only. All towns and cities are urged to organize and prepare in a similar manner.

A deputation of [illegible] is being sent to the Medical Officer of Health of all cities and towns. Further information may be had on application to John W. H. McCullough, M.D., Chairman of Executive, Parliament Buildings, Toronto, telephone Main [illegible]

G. S. WESTON,
Secretary Treasurer

W. D. McPHERSON,
President

J. W. H. McCULLOUGH
Chairman of Executive Committee

Figure 4-6. Call for volunteers
Source: *Kitchener Daily Telegraph*, October 16, 1918.

The role of the newspapers in this pandemic has been noted by a number of authors (Andrews 1977, Belyk and Belyk 1988, Fincher 1989, McGinnis 1976, McGinnis 1977, Weiler 1988) The newspapers published many salutary articles warning of the disease and procedures for caring for the ill. They also printed literally dozens of advertisements for a diverse array of patent medicines. Further "partly to avoid panic, mainly because it was overshadowed by the war, some newspapers tended to downplay the epidemic, just as the country would at first downplay the spread of AIDS" (Fincher, 1989:137-8).

This downplaying of the disease was found in many places. Belyk and Belyk note that in Vancouver the contemporary accounts downplayed the story, tending to refer to the disease only as experienced elsewhere, similar to what was seen in the early stages in Kitchener. Then as the situation grew more serious the

> newspapers appeared more concerned with preventing public panic, than keeping their readers informed. No matter how terrible the situation, no matter how many residents had fallen with the flu or died from pneumonia, local newspapers seemed able to find some spot where the situation was worse. And when it was difficult to convince their readership it was worse elsewhere, newspapers refrained from telling the entire story. Thus, when the number of flu fatalities began to spiral upward in the city, the Nanaimo *Free Press* simply stopped reporting death statistics in its influenza articles (Belyk and Belyk, 1988:49).

Fortunately for this study the Kitchener newspapers never took such drastic action! However, in their efforts to maintain morale, inform their readers and still report what was happening there resulted some interesting juxtapositions of stories.

These attempts at juggling news reporting with morale boosting occasionally had bizarre results. The front page from the *Telegraph* of the 17th October is one example of this. Here one story headlined "Toll of deaths is decreasing" is juxtaposed alongside one bearing the banner "Flu epidemic still unchecked." What does one believe? There seems to be some confusion as to the severity of the situation. The Board of Health meeting that evening decided that people could return to work under these conditions:

> employees of stores and factories who have been
> away on account of illness be not allowed to return
> without a certificate from a physician or registered nurse.
> City of Kitchener, October 17th 1918

This item being reported in the following day's *Telegraph* alongside another positive story headlined "Fewer deaths are reported" and with obituaries relegated to inside pages. Furthermore, the Superintendent at Kitchener-Waterloo Hospital, E. Eisele, telegraphed Dr. J.W.S. McCullough the

Chief Officer, Department of Provincial Secretary, Ontario Provincial Board of Health that

> Forty one cases of Spanish Influenza in residence
> number of calls for admission decreasing
> eight pupil nurses ill
> could use two graduate nurses in hospital
> no other assistance required at the present time.
>
> Provincial Archives of Ontario
> McCullough Spanish Influenza File
> RG 8 1-1-A-1 Box 65 Folder 1

However, another telegraph reaching Dr. McCullough, who was the equivalent of the modern Minister of Health, from Kitchener suggests a different situation. The telegram, from the Mayor of Kitchener, D. Gross, reads:

> Your telegram of 17th statistics unobtainable.
> Epidemic under control but present cases bad
> and need attention, must have four or more nurses
> immediately, wire M.O.H.
>
> Provincial Archives of Ontario
> McCullough Spanish Influenza File
> RG 8 1-1-A-1 Box 65 Folder 1

The 19th October saw the newspapers continuing in this vein. Printing obituaries alongside stories relating that "Many patients are recovering", the readiness of the Allen Theatre for re-opening and the distribution of certificates certifying that people are healthy enough to return to work. (*Telegraph*, 19th October 1918.1) The next day's paper adopted a more sombre note recording the

continuing register of the dead and that during the first nineteen days of the month "Ninety deaths caused by 'flu'." (*Telegraph*, 20th October 1918:1) Hope was not lost though, as "signs of abatement" were there. At least so they hoped. The *Telegraph* reported that these ninety deaths indicated a death rate of 2016 per year or 100 per 1000, whereas 1916 records showed "227 deaths Kitchener that year, being a rate of 10.2 per 1000." A tenfold increase in the death rate due to one basic cause is a most striking indicator of the severity of this pandemic.

Influenza wanes

However, the disease was now beginning to wane. The number of cases was apparently down, the number of deaths was declining (see Figure 4-3) and life was slowly returning to normal. The theatres were preparing for re-opening and classes resumed at St. Jerome's College (*Telegraph*, 20th October 1918:1) However, while some people tried to return the city to some semblance of normality, they were stymied as the Board of Health met on several occasions and decided not to relax the current regulations. (*Telegraph* 22nd October 1918:1, City of Kitchener 25th October 1918, 26th October 1918) It did appear that not only was the crisis past in Kitchener but also across the rest of the province as the *Telegraph* reported the "Flu situation is improving" (22nd October 1918.2) This was followed by the front page news on the 23rd that "Flu epidemic is on the wane in Kitchener"

(*Telegraph*, 23rd October 1918:1), and this time it looks like they were right! Also on the front page of that day's paper were another six obituaries and a short item noting that

> The number of new cases of influenza are reported to be decreasing daily, according to information given by physicians today.

Indeed, the picture improved so much that the headline on the *Telegraph* of the 24th October read "No deaths reported to-day caused by flu." Other stories indicating a return to normality, included one noting that

> only thirty patients remained at the K-W Hospital this morning. A large number of convalescing pneumonia cases have been removed during the last few days.
>
> *Telegraph*, 24th October 1918:1

Another item titled "Want Ban Lifted" recorded that

> There is agitation on the streets to-day for the lifting of the ban on church services before next Sunday. The Board of Health will consider the question tomorrow morning.
>
> *Telegraph*, 24th October 1918:1

However, it was not until the 29th that the Board of Health decided "That all places of amusement, schools and churches be allowed to re-open on 31st inst." (City of Kitchener, 29th October 1918) Indeed, after three weeks of terrific suffering the disease was virtually gone.

All that was left for the newspapers to do was chronicle the last handful of deaths and then summarise the whole ghastly episode into a smattering of articles. Thus the *Telegraph* came to record that in the month of October there had been "149 Deaths in Kitchener . . . About 130 Were Caused by Influenza and Pneumonia." (31st October 1918:1) These 149 deaths exceeded the total for the previous six months of 115. However, the *Kitchener News Record* determines that there were only "One Hundred and Twenty Six Victims of Epidemic During the Month of October, 1918." and that "Never in the City's History Was There a Greater Toll Taken in Human Lives." (31st October 1918:1) Not only does the *News Record* claim that only one hundred and twenty six deaths occurred, it goes on to name the victims and record the dates upon which they died.

Kitchener's factories

Manufacturing has long been of vital importance to the city of Kitchener, and was one area in which those of German origins dominated. Indeed manufacturing almost relied on the German portion of the population; "Germans in Berlin were at the apex of the economic pyramid; they were also overrepresented at its base." (English and McLaughlin, 1983:132) Large numbers of the city's residents have been involved in the manufacture of many goods. In 1911 some 60.2% of the labour force was involved in manufacturing (English and

McLaughlin, 1983:250) (Figure 4-7). Consequently when the city was afflicted by the influenza epidemic of 1918 the factories in the city played a major role in the development of the disease. This role was both negative and positive. Negative in that factories are an ideal place for transmission and contraction of the disease, and positive in that the employers displayed paternalistic attitudes to their workforces that resulted in the creation of company welfare departments that coordinated care for stricken employees.

The negative impact of these centres is obvious when one examines the rolls of the dead, particularly in relation to their occupations, or the occupations of their spouses, parents, etc. It is obvious from even the most cursory glance at this data (Appendix IV) that a significant proportion of those claimed by the disease either worked in a factory of some sort or had familial connections with factories. These included tanneries, textiles (Lang Shirt Co., Star Whitewear), Consolidated Felt, furniture makers (Anthes Furniture, Lippert Furniture), the rubber factories (Kaufmann, Dominion, Merchants) or other places of manufacture (newspapers, carpentry workshops, brewery). Other occupations in which people were exposed to the illness included taxi service, the local school's inspector, hoteliers, salesmen, store clerks and store-keepers. Further

examination of the socio-economic status of the victims is made in the following chapter.

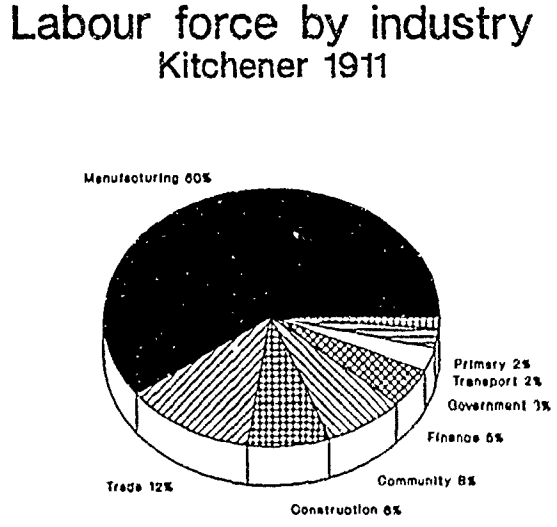


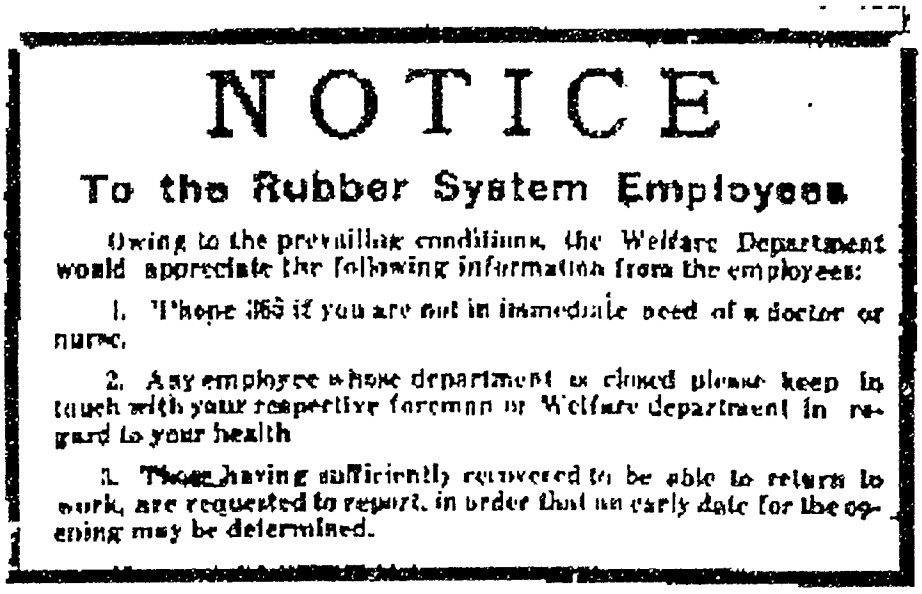
Figure 4-7. Kitchener's workforce by industry.

But it was the factories, especially the rubber producers, that were to play such a role in this crisis. Not only did they serve as a major setting for the transmission of the disease, they also were to take centre stage in attempts to combat the disease. It was the employers who realised very early how important this crisis was. On the 9th October the Hospital Board and the Board of Health met with local politicians and businessmen. Already many cases of influenza were known, in excess of 2429 (*Telegraph*, 10th October 1918:1). E.C. Kabel, manager of Dominion Rubber, suggested that nurses should be organised and transported around the city tending the sick. This was being done by Dominion's Welfare Department, apparently with some success. Furthermore, he suggested that not only should the schools and churches be closed but also that "if closing the factories and all other places of business would help . . . let us close them." (*News-Record*, 10th October 1918:1)

Thus the employers themselves advocated the closing of their businesses for the duration of the epidemic. Weiler suggests that this position was not wholly altruistic as she asserts that "Since Kabel was the manager of Dominion, he would naturally be concerned about the condition of his employees as they affected the interests of the company. Kabel was the most vocal on the closure perhaps because he saw the need to stop the disease as quickly as possible. Even

closing the factories was preferable to having employees die - especially when Kitchener was faced with a lack of skilled labour." (Weiler, 1988:29)

Yet another initiative of the Dominion Rubber System, comprising the Merchants' Rubber Factory, Dominion Tire Factory, Carton Factory, Rubber Machinery Shops and Consolidated Felt, was the expansion of their Welfare Department and the hiring of extra nurses to tend to their ill employees. The company also placed notices informing employees of what to do in case of illness and also what they should do if in condition to work (Figure 4-8).



NOTICE

To the Rubber System Employees

Owing to the prevailing conditions, the Welfare Department would appreciate the following information from the employees:

1. Phone 365 if you are not in immediate need of a doctor or nurse.

2. Any employee whose department is closed please keep in touch with your respective foreman or Welfare department in regard to your health

3. Those having sufficiently recovered to be able to return to work, are requested to report, in order that an early date for the opening may be determined.

Figure 4-8. Dominion Rubber Systems notice.
Source: *Kitchener Daily Telegraph*, October 11, 1918.

Thus the Kitchener manufacturers adopted a paternalistic attitude to their workforce, ensuring their workforce's health to ensure the health of the company. They were willing to take the short term loss of production and business to ensure their longer term corporate health. This was by no means the picture across all of Canada. Vancouver was one Canadian city that underwent a different experience (Andrews 1977, Belyk and Belyk 1988). The sternest opposition to closure of the city "arose from mercantile rather than medical concerns" (Belyk and Belyk, 1988:47) and consequently there "was never any serious consideration given to closing the industrial and commercial heart of the city." (Andrews, 1977:39)

Further examination of the role of the manufacturing and other industry is made in the following chapter. Were those working in the factories overly represented in the death toll? Or were they in fact conspicuous by their relative absence? Was mortality clustered about the factories, was it concentrated in lower class neighbourhoods or was it evenly spread throughout the city both in terms of space and class?

Morbidity

Morbidity data tends to be an underestimation of true morbidity and rarely does it have a temporal or spatial element - there is no intelligence regarding where or when in the time period the cases occurred. Thus little information relating to morbidity exists, and when it does it tends to be anecdotal. In examining the primary sources relating to the 1918 pandemic in Kitchener these difficulties regarding morbidity data are encountered as expected. Basically there is very little and it gives only a brief glimpse into the extent of the disease in the city. The newspapers at times give us scraps of information, often relating to the situation in the city's factories. For example, on the 4th October the *Telegraph* reported that the "Dominion Rubber System . . . have a total of 352 employees laid up with Spanish Influenza." We know that in "1916 Dominion Rubber employed 604 workers" (English and McLaughlin, 1983:134). If we assume that this figure had not changed markedly then we can calculate a level of morbidity of approximately 58%. Other reports in the *Telegraph* also suggest similarly high rates, as were experienced all across the globe. According to reports published on the front page for the 7th October, 1918 several factories were forced shut by absenteeism. Kaufmann Rubber being fairly hard hit with "120 out of 280 on the top flat were on duty" (57% absenteeism), "only one man out of 20 . . . in the mill room" (an astounding 95% absenteeism) and "only 18 out of 60 in the cutting

room were on hand." (70% absenteeism) This situation was repeated across at the Merchants' Rubber Company which "also closed down owing to illness." Large numbers of cases were also noted at Dominion Tire, with 200 ill, and at the H.G. and R. Company where "about 100 cases" were known.

Other indications of the high level of infectivity occasionally appear throughout the month of October. Other stories printed in the *Telegraph* on the 7th documented that some "1735 cases reported by eleven out of the eighteen physicians in the city" and that "at the hospital, which has been closed to visitors, only advanced cases have been taken and they are isolated." Only three days later Mayor Gross was reported as saying that "Reports from some of the physicians on Monday showed 1735 cases and since then I am informed several hundred more cases have been reported. This, I think will give you an idea of the seriousness of the situation." Indeed, and it is worth noting that this was still relatively early in the outbreak (see Figure 4-3). Other stories at the time continued to document the havoc the illness was causing to industry. The newspapers themselves were often short of staff, with absenteeism as high as 75%, and at one stage "over half of the carrier boys are sick" (*Telegraph*, 8th October, 1918). Lang Tanning Company was recorded as having "twenty percent of its men being ill with influenza." (*Telegraph*, 10th October, 1918) while

Dominion Rubber later reported that "at one time, on Tuesday noon, 42.5 per cent of their employees were absent through illness" (*Telegraph*, 11th October, 1918).

A minimum of 2429 cases were known to exist throughout the city by the 10th (*Telegraph*, 10th October 1918:1), at this stage of our population of 104 victims only 24 had died (23%). If the mortality is considered a reasonable and consistent indicator of the prevalence of the disease and thus the assumption that the 2429 cases approximate only 23% of the total morbidity then the estimate of total case numbers is approximately 10 500, a morbidity rate in excess of 53%. This figure is similar to the morbidity reported from Dominion Rubber and is also similar to the rates found in the literature for other cities (Andrews 1977, Crosby 1989, Heagerty 1928).

However, this is the entire extent of morbidity data and it allows us little scope for analysis. In order to investigate the nature of this pandemic further it is necessary to undertake an examination of mortality data. Both types of data contribute to our understanding of the disease.

Mortality

One aspect of the 1918-19 pandemic that was somewhat unusual, for influenza, was the ages of the victims. Influenza typically claims the young and the aged. While both of these groups certainly did suffer (Figure 4-9), especially the children aged 1-10, the peak age group in terms of fatalities was the 21-30 age group. Crosby (1976) discusses the normal state of influenza mortality and the somewhat unusual mortality of the 1918 pandemic.

> Influenza and pneumonia, when they kill, usually kill those of two extremes of life, the very young and the old. The curve of influenza and pneumonia mortality relative to age for 1917 was . . . a crude U, high at both ends and low in the middle, as common experience suggests it should be. When an influenza epidemic or pandemic strikes, both the influenza and pneumonia mortality rates rise, but the shape of the curve remains approximately the same. Influenza and its complications still kill the young and the old more readily than those in the prime years of life. But when a curve is plotted for the incidence of flu and pneumonia deaths according to age for one of the United States cities that had a higher than normal number of such deaths in the spring of 1918 . . . the resulting curve is not a U, but a crude W with its highest point in the middle, where both science and common sense declare it should not be. Analysis of the death certificate files for other American cities . . . - New York City and Lowell, Massachusetts in the Northeast, Birmingham in the Southeast, San Francisco and Seattle on the Pacific Coast, and Minneapolis in the North Central - results in the same spike in the middle, usually in the 21-29-years-of-age column. (Crosby, 1976:21)

This is exactly the case found when one examines the records for Kitchener in October 1918 (Figure 4-9). Crosby goes on to note that this pattern was widely experienced, including in the cities of London and Paris (Crosby, 1976:27) as well as Philadelphia (Crosby, 1976:86) and all the other American cities already mentioned. This pattern has been found in virtually all populations scholars have scrutinised, (Andrews 1977, Belyk and Belyk 1988, Beveridge 1977, Kilbourne 1987, McGinnis 1977, Monto 1987, Pyle 1986, Stuart-Harris *et al.* 1985) including the Canadian cities of Toronto, Montreal, Kitchener, Winnipeg, Calgary and Vancouver.

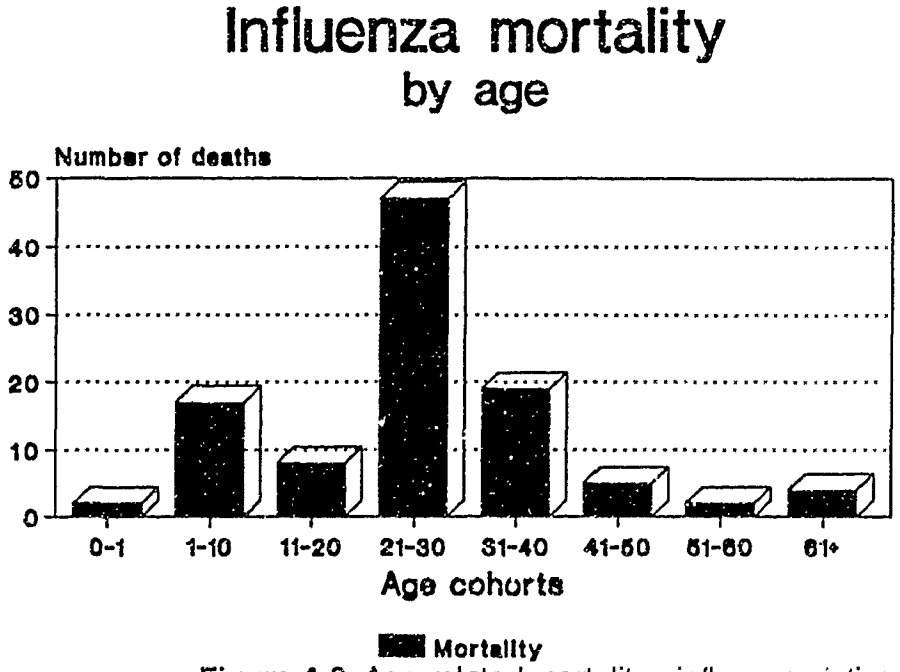


Figure 4-9. Age-related mortality - influenza victims, Kitchener 1918.

Kitchener's experience of influenza-related mortality in the pandemic then is quite typical of the pandemic as it occurred elsewhere. This pattern of death by age was recognised at the time as the newspaper articles of the time commented that "Flu Victims Mostly Young - Sixty-Five Percent of Deaths in Ontario Are of People Between 20 and 30." (*Telegraph*, 23rd October 1918:8) and that the "Death Toll Among Young Was Heavy - Over Eighty of Victims of Influenza Were Under 30 Years of Age." (*Telegraph*, 1st November 1918:10) This was true for both sexes, as can be seen in Figure 4-10. However, it is interesting to note that male mortality is higher, not just in total numbers but also in those particularly significant age groups of 21-to-30 and 31-to-40. (Hope-Simpson, 1992:27, Pyle, 1986:50) Male and female mortality in other age cohorts was similar but in those two groups with the largest number of deaths males died in appreciably higher numbers, this was as has been noted earlier a "remarkable feature in 1918 was the high mortality among young adults, especially males." (Hope-Simpson, 1992.27) However, no convincing explanation for either of these idiosyncrasies of the 1918 pandemic, the high young adult mortality and the propensity for male deaths, has been forthcoming.

In Kitchener the young males who succumbed came from all socio-economic groups, judging upon their occupations. While many were employed in the

factories or in construction, typically the employers of many young men, others earned their living in the stores or offices. Thus from this study come no revelations for this unusual pattern of mortality.

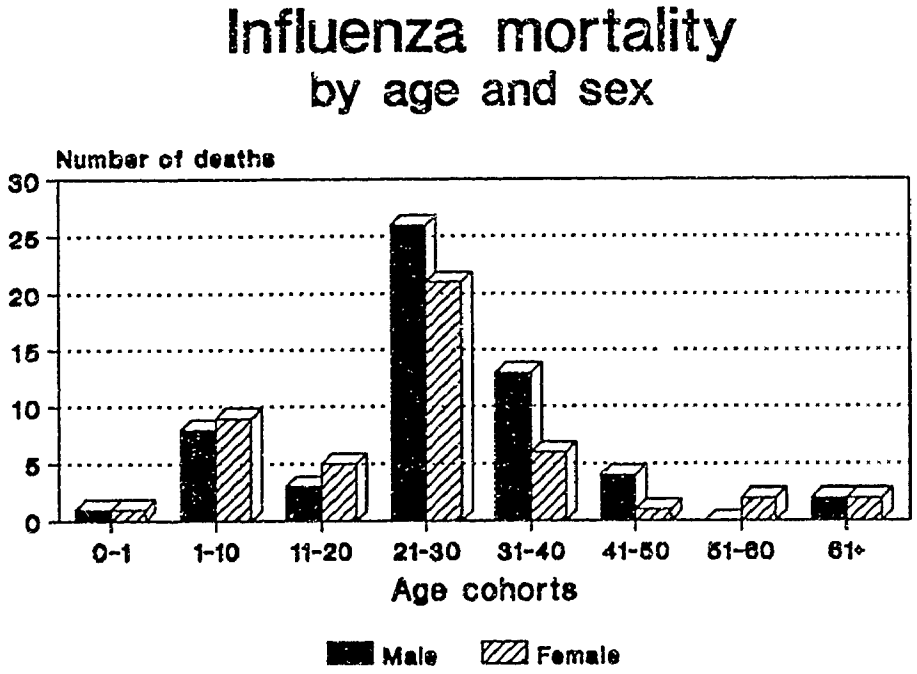


Figure 4-10. Age-sex mortality for influenza victims, Kitchener 1918..

All across Canada, as across the entire globe, the disease caused massive morbidity, striking at least half the world's population, and excessive mortality, killing as many as 50 million. Conservative estimates for Canada claim that "at least one-sixth of the entire population was attacked and that 30,000 of these died. In later years the mortality estimate has been revised upwards to around

50,000, with a consequent increase in the morbidity estimate. The Canadian death rate per 100,000 for the years 1917, 1918 and 1919 shows a jump due to the epidemic: 12.7, 15.9 and 13.7, respectively." (McGinnis, 1977:132)

From coast to coast there were was great suffering. In New Brunswick at least 1,394 people died (Heagerty, 1928:220), while in Quebec there were some 530 704 cases reported and of these 13 880 were fatal (Heagerty, 1928:219), a 2.6% case fatality rate. However, such rates must be treated with caution as the reporting of cases is certain to be significantly understated. The disease was particularly severe in Montreal with 17 252 cases reported and 3 028 deaths, (Heagerty, 1928:216) an astounding 17% case fatality. However, it is thought that Montreal had at least 100 000 cases (Heagerty, 1928:216), a case fatality rate of 3%, still somewhat high. Once again the estimates of morbidity would appear rather conservative. Ontario was also heavily struck, with estimates of "at least 300,000 cases of the disease and the total number of deaths . . . was 8,705" (Heagerty, 1928:219), a case fatality rate of 2.9%. Toronto had 2 284 of these deaths, with a death rate of 14.3 per 1000 (Andrews, 1977:21). Of these 8,705 Ontarian deaths some 127 were in Kitchener. The city has seen its death rate climb markedly. The toll for the previous six months from all causes was only 115 with a crude death rate of 96.96 deaths per 100 000 per month. Now in one

month influenza had claimed 127 victims. This gives a crude cause of death rate of 642.85 per 100 000 per month, nearly seven times greater. Furthermore, if one assumes a morbidity rate of 50%, a reasonable assumption based upon the experience elsewhere and the limited information we have relating to morbidity in Kitchener, the case fatality rate of approximately 1.3% is similar to that found in many localities (Beveridge 1977, Crosby 1989, Kilbourne 1987, Stuart-Harris *et al.* 1985).

Western Canada was by no means spared with some 3 906 deaths in Saskatchewan (Heagerty, 1928:220) and Alberta witnessed an estimated 38 000 cases "of which over 4,000 died." (McGinnis, 1976:1) Calgary apparently escaped the wrath of the disease with only 341 deaths, however, "non-statistical data indicates that the city's actual toll was considerably higher." (McGinnis, 1976:1) Winnipeg suffered some 1 021 deaths, with a death rate of 16.7 per 1000 (Andrews, 1977:21). Vancouver was by no means as fortunate, where by "a conservative estimate, the epidemic sickened 30,000 and killed 900 of a population of about 100,000." (Andrews, 1977:27) Table 4-1 summarises the impact of the disease across a number of Canadian cities.

Influenza Deaths

City	Deaths	Death rate per 1000 of population per annum
Toronto	2 284	14.3
Winnipeg	1 021	16.7
Vancouver	795	23.3
Kitchener	127	6.42[a]

[a] Crude cause-specific death rate per 1000 per month

Table 4-1. Influenza deaths and crude death rates in Canadian cities.
Adapted from Andrews (1977:21)

Chapter 5. The geography of influenza in Kitchener.

A desperate disease requires a dangerous remedy.
Guy Fawkes.

In the preceding chapter I described the influenza pandemic as it struck Kitchener. Now I will analyse the pattern of the disease and produce a sequence of maps depicting the spread of the disease, as expressed by the associated mortality, through the city. To do this I will first make some comments on epidemics, followed by a discussion of disease diffusion and mapping of disease Following this will be the sequence of maps and discussion of a possible barrier to diffusion - the imposition of quarantine. In the final portion of the chapter some analysis of the spatial patterns of mortality associated with the city wards, socio-economic factors and age will be presented.

Mention has already been made of the difficulty in obtaining detailed data regarding disease morbidity, particularly for historical outbreaks. As a consequence of this, those who study disease make extensive use of mortality

data in order to learn more about the disease under examination. Furthermore, as Crosby so neatly puts it:

> Statistics on mortality were and always are more dependable than those on illness. Death can be diagnosed correctly by anyone, and the cause, in the case of death by disease, is usually that disease whose symptoms were most grossly apparent in the patient on the eve of his demise. (Crosby, 1989:204)

A number of the problems pertaining to the data were discussed earlier. Patterson (1986.8-9) lucidly delineates the problems of historical data relating to influenza in his study of pandemic influenza in the period 1700 to 1900.

Mapping the pandemic in Kitchener

The examination of disease from a geographical perspective has a long tradition of utilising cartographic display, dating back to John Snow's famous dot map of cholera victims around London's Broad Street pump. It is in this tradition that uniform dot maps have been employed as the primary cartographic vehicle for examining the geography of the 1918 influenza pandemic in Kitchener.

Production of these uniform dot maps required at least one question answered. How often should the progress of the disease be mapped? Daily? Weekly? Daily is impractical and of questionable value. Thus the selection of

periodicity for these maps was based upon the incubation period of the disease. As the influenza of 1918 had an incubation period of 2 to 4 days these maps were then produced for each 4 day period in October (Figures 5-1 to 5-8), with the exception of the first map (Figure 5-1) which refers only to the first three days of October 1918. Thus each map displays the deaths that resulted from those cases that were contracted in the previous period. Also displayed, with the map for each period, is a cumulative map (Figures 5-2a to 5-8a), displaying all the deaths up to and including that time.

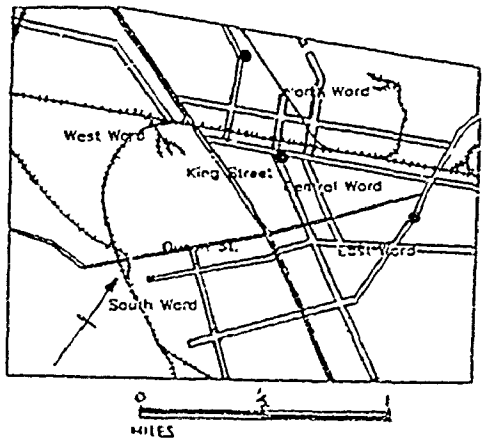


Figure 5-1. Influenza deaths - October 1st to 3rd.

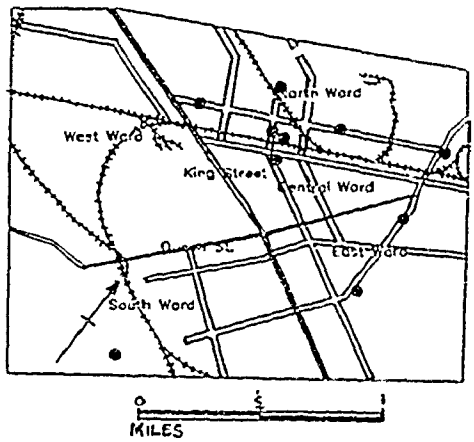


Figure 5-2. Influenza deaths - October 4th to 7th.

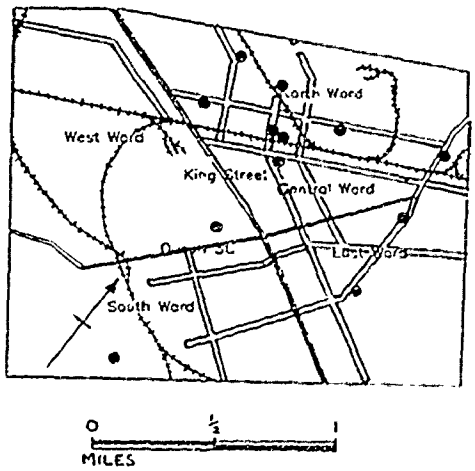


Figure 5-2a. Cumulative influenza deaths - October 1st to 7th.

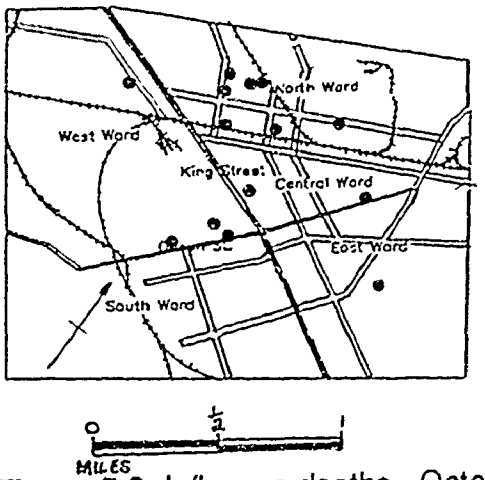


Figure 5-3. Influenza deaths - October 8th to 11th.

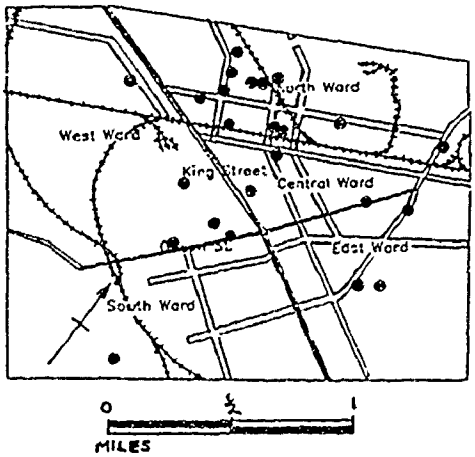


Figure 5-3a. Cumulative influenza deaths - October 1st to 11th.

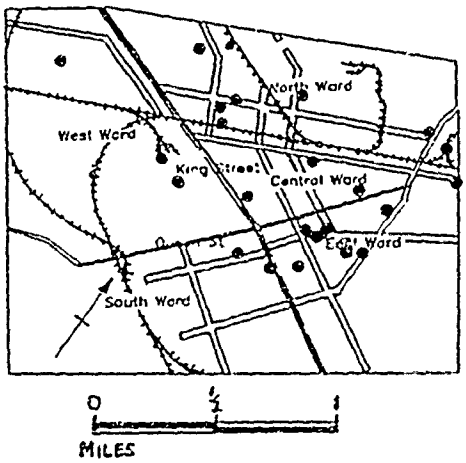


Figure 5-4. Influenza deaths - October 12th to 15th.

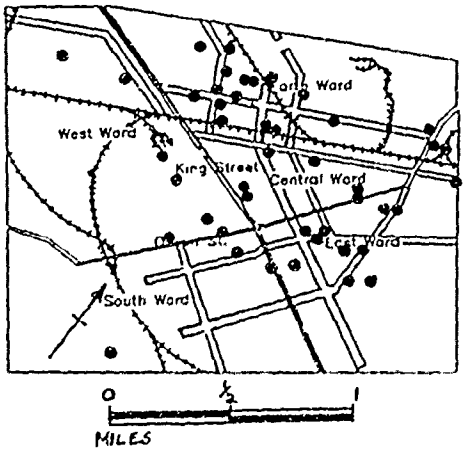


Figure 5-4a. Cumulative influenza deaths - October 1st to 15th.

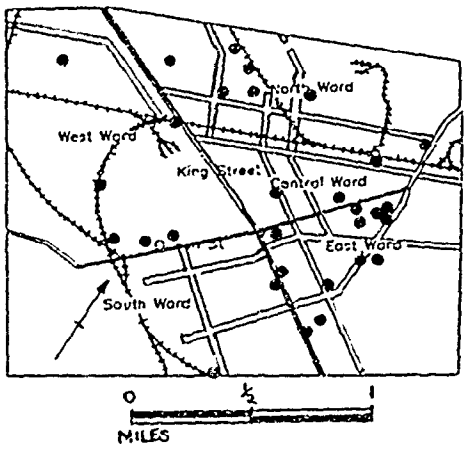


Figure 5-5. Influenza deaths - October 16th to 19th.

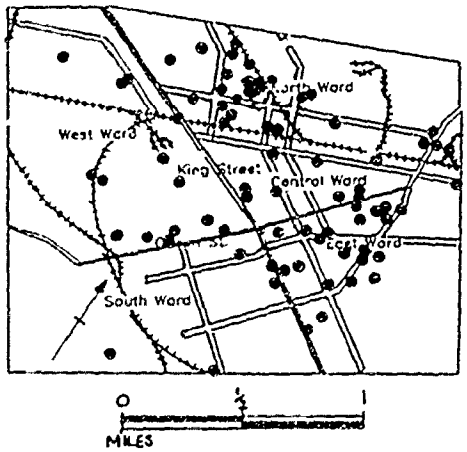


Figure 5-5a. Cumulative influenza deaths - October 1st to 19th.

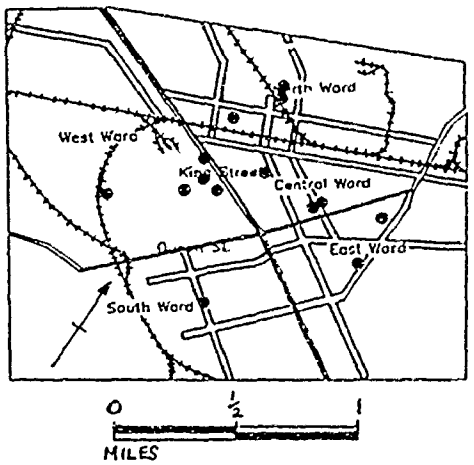


Figure 5-6. Influenza deaths - October 20th to 23rd.

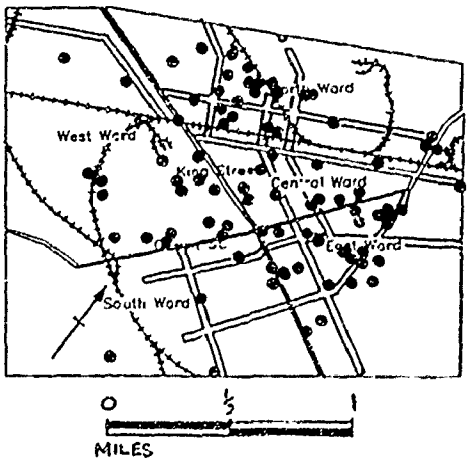


Figure 5-6a. Cumulative influenza deaths - October 1st to 23rd.

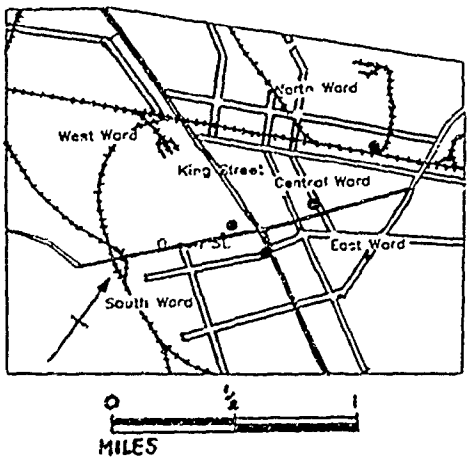


Figure 5-7. Influenza deaths - October 24th to 27th.

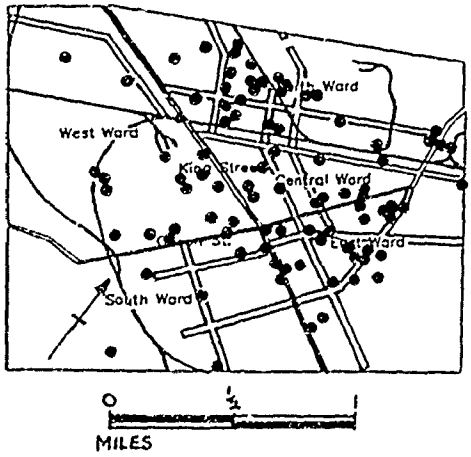


Figure 5-7a. Cumulative influenza deaths - October 1st to 27th.

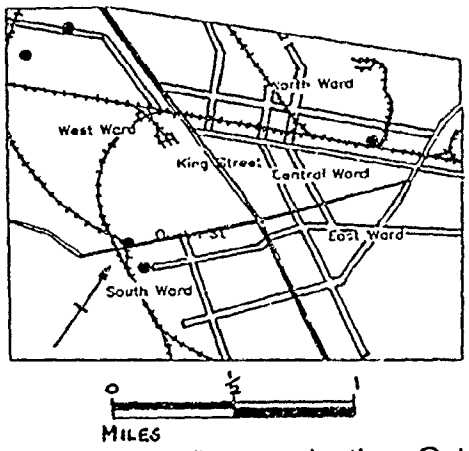


Figure 5-8. Influenza deaths - October 28th to 31st.

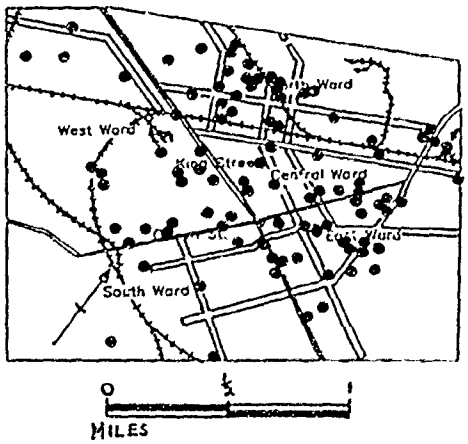


Figure 5-8a. Cumulative influenza deaths - October 1st to 31st.

These maps complement the epidemic curve produced in Chapter 4, and reproduced here (Figure 5-9) in that the peaks and troughs of the curve are reflected in the number of cases appearing as the sequence progresses. Figures 5-1 and 5-2 show the rise of the disease, Figures 5-4 and 5-5 show the fatalities at their peak and Figures 5-7 and 5-8 depict the waning of the epidemic.

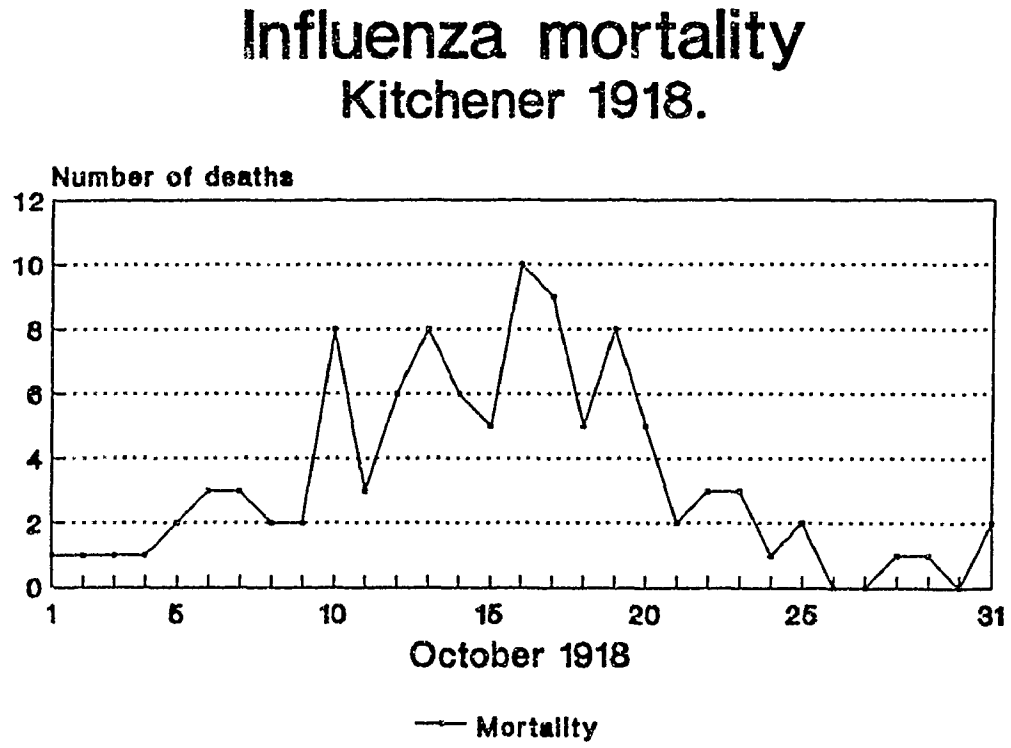


Figure 5-9. Epidemic curve.

The pattern of fatalities indicates where the disease was prevalent just days before. As the disease moves through it leaves a trail of death in its wake, the

deaths occurring 2-4 days after infection. It is this trail that is appearing in these maps. With such a contagious disease as influenza it is expected that quite often in reviewing the maps of mortality that clusters of deaths emerge. Indeed this appears to be true as early as the 3rd of October, (Figure 5-1) when two of the 4 deaths occurred at the same location, the Station Hotel (the dots overlay one another on the map). Initially the fatalities display apparently binodal origins with deaths appearing to occur slightly more frequently in the North and East Wards of the city (Figure 5-2 and 5-2a). From these origins the disease goes on to form clusters across the city. Yet more deaths occurred in the North Ward over the next few days (Figure 5-3). The disease appears concentrated in the North Ward in the early stages of the pandemic (Figure 5-3a).

The period of the 12th to the 15th of October (Figures 5-4 and 5-4a) sees the disease penetrating the entire city, with fatalities in all the city wards. Now the northern and eastern sectors are continuing to display concentrations of deaths, but the disease is also prevalent elsewhere (Figure 5-4a). These figures and the next pair, covering the 16th to the 19th (Figures 5-5 and 5-5a), show the epidemic at its peak (Figure 5-9) with deaths occurring throughout the city. The clusters are becoming denser, particularly in the east of the city. A "neighbourhood

effect", typical of diseases spread by person-to-person transmission, is likely to be playing a major role in the spread of the disease.

October 20th to 23rd (Figure 5-6) indicates that the disease was continuing to spread through the West and Central Wards while fatalities declined in the North and East Wards. The following maps (Figures 5-7 and 5-8) depict the last throes of the disease as it wanes as the month draws to a close (Figure 5-9).

Thus it is apparent that the disease took hold across the city. The neighbourhood effect, the emergence of cases close to one another, is evident in these maps, notably the 4-day period maps (Figures 5-1 to 5-8). The cumulative maps (Figures 5-1a to 5-8a) show how the disease very quickly disseminated itself throughout the city with concentrations apparent in the North and East Wards and a corridor of cases stretching from the north of the city through the Central Ward out through the eastern portion of the city. Also evident is a corridor of cases stretching from the Central Ward west into the West Ward (Figure 5-8a).

Isoline mapping has been extensively used in the geographical study of disease. This has included the mapping of isomorts, contours of death, to examine spatial variations in mortality or the production of isoline maps to chart

the spread of disease (Figure 5-10) over space and through time. Pioneered by Learmonth and Nichols (1965) the technique has gained widespread acceptance and has been used for depicting the spread of a number of historic outbreaks of disease (Carpentier 1962, Patterson 1986, Pyle 1986). Here (Figure 5-10) the 4-day periods used to disaggregate the data for the dot maps were now used to delineate contours. The process is detailed in Appendix VI. The resulting pattern of isochrones (Figure 5-10) is not altogether clear. However, it is recognisable that the outbreak appears to have occurred first in the northern parts of the city, with another early centre being to the east of the city, and another to the southwest. From these the disease radiated out. Later, for periods five, six and seven from October 16th to October 27, contraction of the disease appears to be evident in the east and southwest. Thus we have the multinodal origins that the literature suggests for infectious respiratory disease (Meade *et al.*, 1988:241) The wave progression of the disease, as it swept across the city, is more clearly seen here than in the dot maps.

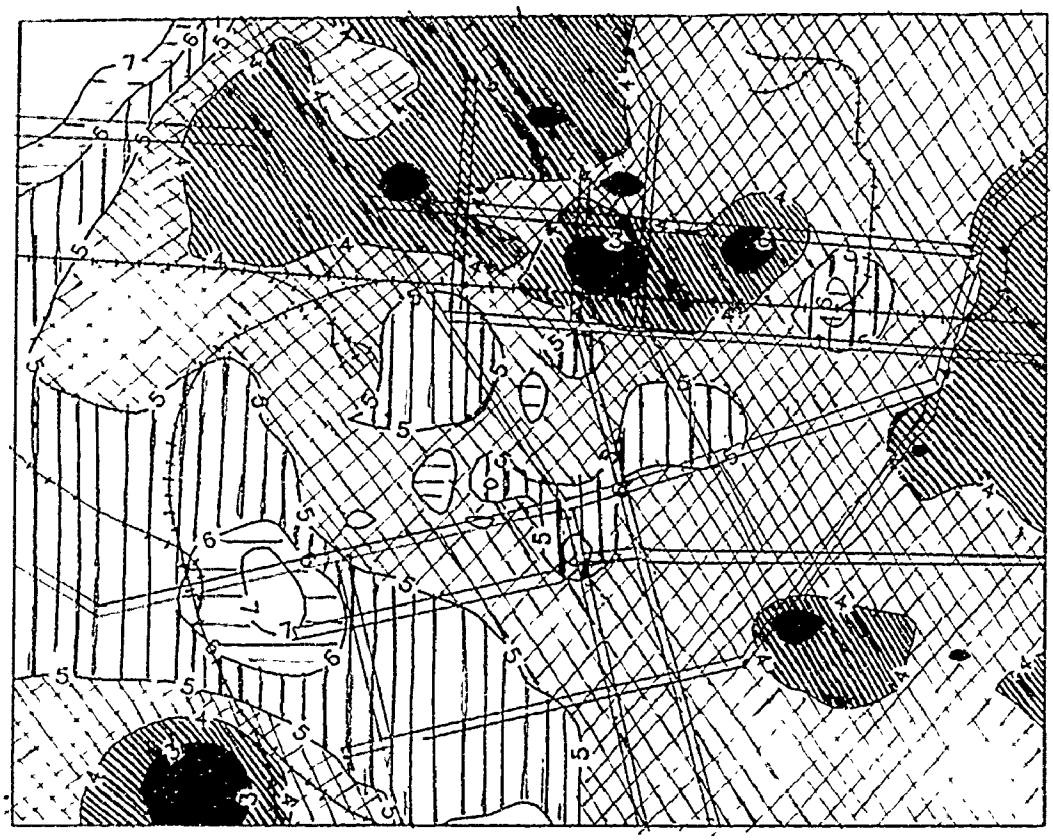


Figure 5-10. Isochrone map of influenza mortality by 4-day period, Kitchener 1918.
N.B. Integers refer to 4 day periods starting October 8th (3) through to October 31st (8).

Effect of quarantine

The Kitchener Board of Health imposed a semi-state of quarantine on the 7th October, 1918 by decreeing that all schools, churches and theatres be closed, as was described in the previous chapter. This quarantine was later expanded to include all places of entertainment, such as bars. Quarantine, logically, is a

potential barrier to the continued diffusion of a disease. But what effect did this proclamation have? Apparently very little, judging by the mortality data gathered here. As was discussed in the previous chapter, the mortality curve (Figure 5-9) shows that peaks of the disease occurred on the 10th, 13th and 16th and considering that the influenza of 1918 displayed an incubation period of 2 to 4 days it is logical to conclude that quarantine had little effect in stopping the disease. This view is reinforced when examining the various maps presented here (Figures 5-1 to 5-8a, 5-10) where the disease is very much seen to continue spreading throughout the city over a period of two to three weeks and did not wane until towards the end of the month, in keeping with the experience in many North American cities (Galishoff, 1969 in Marks and Beatty, 1976:274). Thus it is apparent that the quarantine imposed upon Kitchener was such a permeable barrier that is had a negligible effect and the disease ran its course, waning only when the susceptible population was too small to support the continued spread.

Wards

Kitchener in 1918 was divided into five small administrative areas called wards. These were small areas largely delineated along King and Queen Streets, none larger than approximately a square mile. From the dot maps and the

isomort maps it is suggested that influenza may have appeared in some wards earlier than others and then spread to others. Indeed in examining the date of fatalities by ward (Table 5-1) it is apparent that it struck earlier in some areas. From this the impression that the disease existed in the North and East Wards first and was found later in the West and South Wards is borne out. However, it is not correct to say that the disease started in the North and East Wards and then relocated to the rest of the city as fatalities were recorded in these wards through much of the month (Figure 5-11). Thus the disease was first found in certain areas but was prevalent throughout the entire city for much of the month of October 1918. Furthermore, when the wards were re-ordered by the mean date of fatality, assigned values 1 to 5 with 1 being the ward with the lowest mean date, and correlated with the date variable the correlation coefficient derived was only .3016, suggesting an insignificant level of correlation between the wards and the date of death.

Ward	Date		
	Average	Min.	Max.
North	12.8	1	25
East	14.3	4	21
Central	15.1	2	23
West	17.1	8	31
South	18.4	5	28
Total	15.06	1	31

Table 5-1. Date of fatalities by ward.

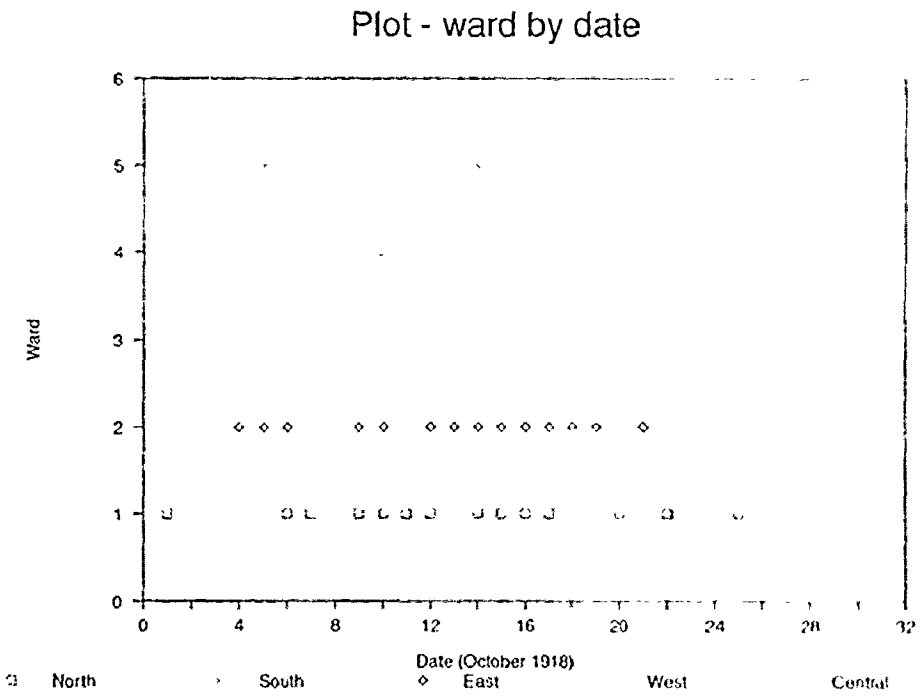


Figure 5-11. Plot of city ward by date of fatality.

However, this slight temporal variation between the wards is not the only difference. Interestingly there are noticeable variations in the volume of fatalities between the wards, particularly the South Ward, as is evident in the maps (Figures 5-1 to 5-8a). Indeed when one compares the crude death rates between the wards (Table 5-2) the South Ward stands out from the other wards. Not only did the fewest deaths occur in the South Ward but, more importantly, the crude death rate is several times lower than that of any of the other wards.

Ward	No. of cases	Popn.	Rate per 1000 population
North	26	4122	6.308
West	26	4215	6.168
Central	15	2455	6.110
East	29	5460	5.311
South	8	3515	2.276
Total	127[a]	19767	6.425

Table 5-2. Crude death rate by ward.
[a] Column sum does not equal total deaths as not all deaths are known by location.

Socio-economic factors

A major concern throughout this study has been the possible role of socio-economic differentiation among the victims of the pandemic. Socio-economic factors can greatly influence people's health. This influence can include the ability to obtain medical help, the knowledge of what help exists, the quality of the dwelling and the pre-existing state of health before disease. The poorer classes in Kitchener are likely to have had a lesser general state of health and thus more likely to suffer when attacked by the influeza virus. Influenza, theoretically, should strike all strata of the population equally, showing complete disregard for status. Even in this epidemic the literature recognises no socio-economic differentiation among the victims (Beveridge 1977, Braithwaite

1953, Collier 1974, Crosby 1989, Fincher 1989, Gallagher 1969, Grist 1979, Noll 1989, Pettigrew 1983, Starr 1976) . However, was this the case in Kitchener in October 1918? Was there any relationship between the socio-economic status of the victims and mortality in this pandemic? Did those who died tend to come from any particular socio-economic class? Were certain occupations and/or workplaces more hazardous than others? Were the deceased evenly spread throughout the community or were certain groups or areas overrepresented? There are many aspects of the human condition relating to socio-economics to be examined here, including housing, occupation, and income but the limitations of extant data impinge heavily. Little data on housing conditions were available and only partial data upon the socio-economic conditions, thus limiting such considerations.

Density

Could the variation in crude death rate by ward be a function of density? From contemporary maps and the Assessment Rolls it is known that the large South ward has a large number of empty lots. The ratio of people to lots, a potential surrogate for density, is substantially lower in this ward than elsewhere (Table 5-3). Further, the Central Ward with the smallest population (Table 5-2) has the highest crude death rate (Table 5-2) and the highest population to

number of lots ratio (Table 5-3). Curson and McCracken remark that "population density tends to be a more important factor in infectious disease incidence than in chronic degenerative conditions" (1989:3.7) and Crosby, as noted in Chapter 3, observed that "[S]ometimes there was a discernable correlation between flu, pneumonic complications and crowded living conditions" (1989:228). However, it is important to note that this potential surrogate measure for density refers to the density across an entire ward and in no way can be said to indicate density at the individual household level. Futher, population density is an important factor in the diffusion of diseases that require close contact for transmission. If population density data were available this could allow for simulation of the diffusion of the disease, but this data is not available at the detailed level, e.g. household level, that would make modelling feasible.

Ward	No. of lots	Popn.	Popn./No. of lots
North	4122	1830	2.25
West	4215	2148	1.96
Central	2455	976	2.52
East	5460	2577	2.12
South	3515	2602	1.35
Total	19767	10133	1.95

Table 5-3. Population and lot data by ward.

Housing

Density is just one factor of the housing environment and one that may have a socio-economic component. Already touched upon above was the possible role of density in the lower incidence of fatalities in the South Ward. Housing is of crucial importance in questions of health as

> housing is the milieu in which most individuals spend the greatest part of their lives and has long been considered to have an important impact on human health . . . while popularly accepted as a significant factor it is often very difficult to isolate the adverse affects of poor housing on health from the effects of other social and economic variables (e.g. social class, poverty). The multifaceted nature of the housing environment also makes it difficult to identify the effects of individual housing factors (e.g. state of repair, size, facilities, design, crowding and privacy). Investigation of the relationship is further complicated by the possible operation of a reverse causal path (i.e. - poor health may result in low income and impel residence in cheap, low quality housing). (Curson and McCracken, 1989:3 11)

In this study no consideration has been given to the housing environment. No data pertaining to the size, design or quality of the dwellings were found. Consequently no analysis of mortality with regard to housing has been made. The implications of this for future research as discussed in the concluding chapter.

Socio-economic factors and mortality

Curson and McCracken, as cited above, noted that housing was just part of the socio-economic factors that could influence health status. Furthermore, they state that

> mortality levels are influenced by socio-economic factors that are differentially distributed by social class . . . social class morbidity and mortality differentials take on geographical expression and significance through the differing spatial distribution of social class groups. (1989:3.13)

Within social class Curson and McCracken recognise three fundamental socio-economic differentiators of class: income, occupation and education. Each of these have great impact upon human health:

> Income . . .
> - influences the material standards of living affecting health status (e.g. housing quality, nutritional level) attainable . . .
>
> Education . . .
> - through its relationship with income and occupation.
> - influences health knowledge, attitudes and behaviour:
>
> e.g.- attentiveness to personal preventative measures
> - recognition of symptoms
> - preparedness to seek treatment . . .
>
> Occupation . . .
> - exposures to hazards in the work environment . . .
>
> Biological hazards
> - e.g. infections and parasitic agents
>
> (1989:3.13-14)

Indeed some authors have taken these concepts of social class and their impact upon health and formulated societal models of disease in which

> The mortality structure of a society is determined primarily by the nature of the social organisation . . . from the Marxist point of view, the class structure of society and the resulting pattern of social relations determines the health of the population. The process of capital accumulation becomes the disease-producing force . . . (Cooper and Schatzkin, 1982:459-460)

But do class considerations play any role in determining mortality in the 1918 influenza pandemic? Is there a socio-economic differentiation among the victims? One approach to this question is to examine the local Assessment Rolls, which give information regarding the economic status of residents in terms of assessed property value and assessed taxes. When histograms of this data, unfortunately incomplete, are plotted (Figures 5-12 and 5-13) and the data examined (Tables 5-4 and 5-5) there is a suggestion that the victims did tend to be of lower economic status. The majority of values are to the lower extremes of the histograms and the median and mode values are notably lower than the mean values.

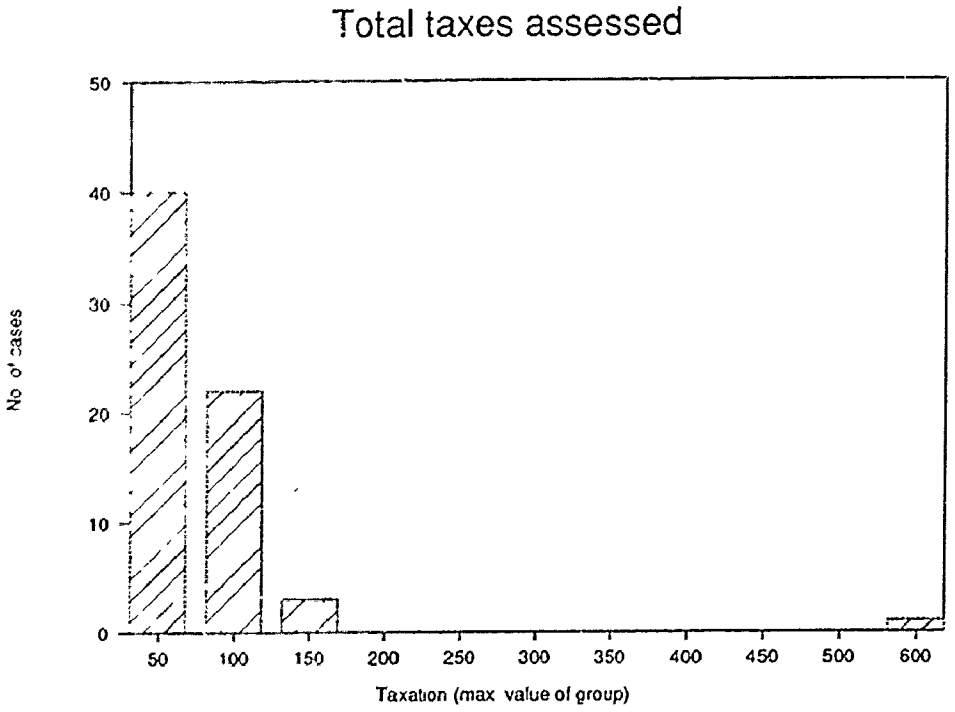


Figure 5-12. Assessed taxation values for influenza victims, Kitchener 1918.

Assessed taxation

Mean	58.627	
Median	42.89	
Mode	32.72	
Range	592.66	
Minimum	3.57	
Maximum	596.23	
Valid cases	66	Missing cases 38

Table 5-4. Total taxes assessed - influenza victims, Kitchener 1918.

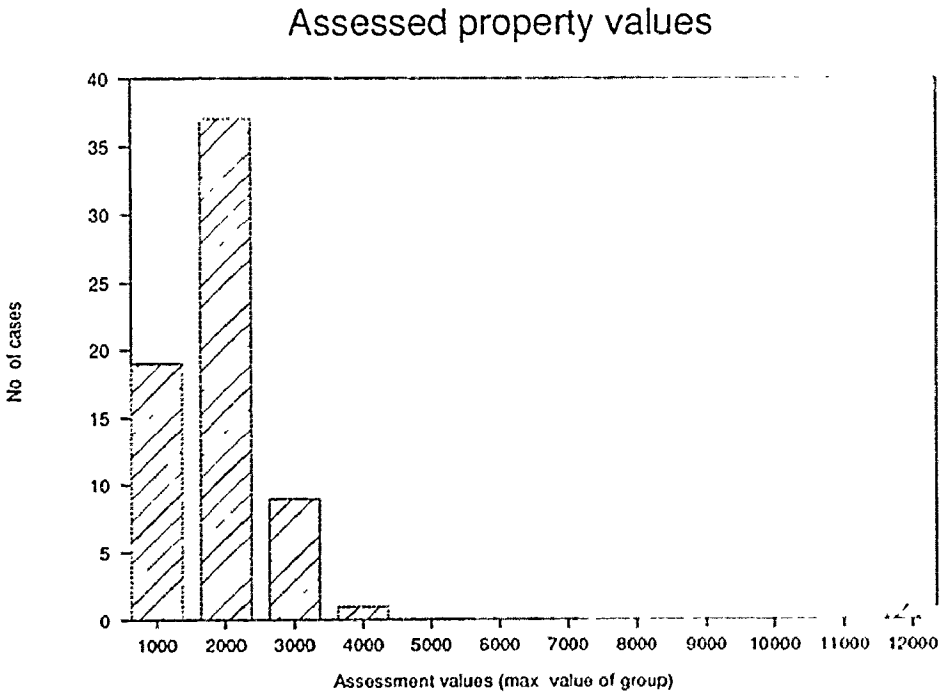


Figure 5-13. Total assessed property values - influenza victims, Kitchener 1918.

Total assessed property value.

Mean	1441.134	
Median	1140.00	
Mode	700.00	
Range	11005.00	
Minimum	120.00	
Maximum	11125.00	
Valid cases	67	Missing cases 37

Table 5-5. Total assessed property value - influenza vitctims, Kitchener 1918.

When examined on a ward basis (Table 5-6) the taxation and assessment data suggest that some of those wards with the highest numbers of fatalities are those with the lowest mean values for assessed property values and assessed taxation, notably the North and West Wards. This suggests that there may indeed be a socio-economic factor in some of the deaths recorded in Kitchener. However, it should also be recognised that the South Ward which recorded the fewest deaths at the lowest rate also has relatively low values for these measures. Thus, this issue of economic association with mortality is unclear.

	Total		Influenza victims only						
Ward	taxable property	Mean	Age mean	Taxes assessed Mean	Min	Max	Assessed value Mean	Min	Max
Total	10658159	538.65	26.6	58.63	3.57	596.23	1441.13	120	11125
N	1468818	356.34	25.8	46.21	14.87	80.52	1266.50	500	2112
W	2450550	578.64	27.6	53.97	5.36	131.41	1167.90	180	2540
C	2146035	874.56	28.7	135.87	3.57	596.23	2712.40	120	11125
E	2758124	505.15	25.9	57.09	20.87	86.64	1493.90	700	2610
S	1833632	521.66	25.1	44.41	7.02	95.44	1390.80	236	3047

Table 5-6. Tax and assessment data by ward, Kitchener 1918.

Manufacturing

Occupation is a major component in determining income, socio-economic status and social class. The importance of manufacturing in providing

employment in 1918 Kitchener has already been discussed. This leads to questions of how did occupations, particularly manufacturing-related positions, impact upon influenza mortality? Were the factories prime locations for the contraction and transmission of the disease thus leading to increased morbidity and mortality among manufacturing workers? Or did the employee care programs initiated, notably by the Dominion Rubber System, mitigate the disease and contribute to a lower than expected mortality experience for employees?

The division of the workforce into certain industries is known, from census data (English and McLaughlin 1983:250-1). By taking the known occupations of the victims or their spouse or parent(s) and then classifying into similar categories it is possible to compare the division of the victims' (or the victims' household breadwinner) by industry with that of the total labour force (Table 5-7.) From this it is apparent that those employed in manufacturing may have died at a lesser rate than could be expected, as they comprise only 44.32% of those who died (for whom occupation was known) whereas manufacturing employed 57.0% of the 1921 workforce. Another sector to suggest mortality at rates lower than may be expected was the community, business and personal service sector. Sectors that apparently had greater mortality include construction,

transportation and communications, trade (including retail), finance, insurance, real estate and government employees. Note that most of these are areas in which workers tend to have greater contact with people. However, a number of factors should be noted, including the small size of the population for whom occupational data was obtained and the likelihood of under-reporting. Nevertheless it is apparent that many of those succumbing to the disease came from working class households. Again there is the suggestion that socio-ecoromic factors could indeed have had an influence on the disease outcome.

Occupation category	No. of deaths	%age of total	Percentage workforce in industry [a]	Critical value[1]
Manufacturing	39	44.32	57	-2.40*
Primary	0	0	0.7	—[2]
Construction	12	13.64	6.0	3.02*
Transport and communications	12	13.64	5.0	3.72*
Trade	14	15.91	10.0	1.85
Finance	4	4.55	3.0	—[2]
Community	1	1.14	13.0	—[2]
Government	6	6.82	3.0	0.78
Other	0	0	3.0	—[2]

[1] Test for difference of proportions.
[2] Group too small for test.
* Significant at 0.05 level.

Table 5-7. Labour force and deaths by occupation/industry.

[a] 1921. Source: English and McLaughlin (1983:250-1)

But what of the spatial arrangement of those who died in the different occupations? Manufacturing, as has been seen, was of major importance in Kitchener at the time. Thus, to examine the spatial variation between occupations the population was split into two groups: those employed in manufacturing and those employed elsewhere. These two groups were then mapped (Figures 5-14 and 5-15). From these it is readily apparent that in the North Ward the dead tended to work in the factories. In the West Ward the fatalities are evenly split between manufacturing and non-manufacturing employment while in the remaining wards, east, central and south, tended to be employed in the non-manufacturing sectors. But what is the significance of these patterns? How does one interpret them? These patterns would appear to be most significantly related to the location of the workplace. This is evident when one examines the location of the factories at that time (Figure 5-16). The factories are largely found along the boundaries of the wards, notably between the North, Central and West Wards. This pattern reflects that found in Figure 5-14, and suggests short journeys to work, as a commentator noted at the time when he wrote:

> Berlin is a town of smokestacks . . . Once off the train, you are fair in the midst of tall chimneys . . . The workers live among the factories. The advantage of this is little or no cost of transportation. The disadvantage is smoke which, no matter where the wind is, floats over the houses. (Bridle, 1906)

Thus it would appear that spatial variation in the pattern of mortality is based more upon the location of the place of employment than anything else.

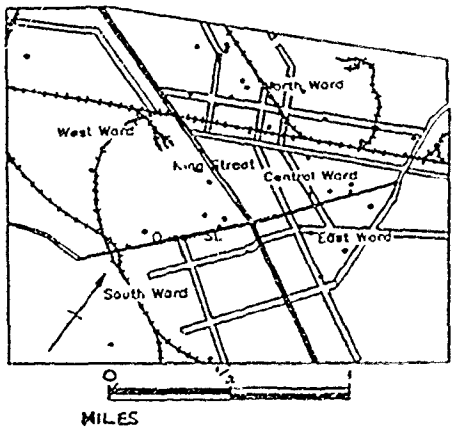


Figure 5-14. Victims employed in manufacturing.

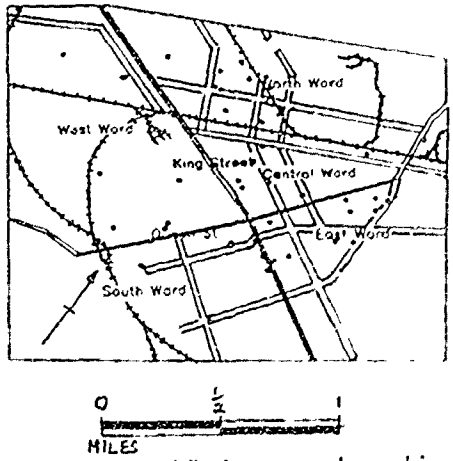


Figure 5-15. Victims employed in non-manufacturing positions.

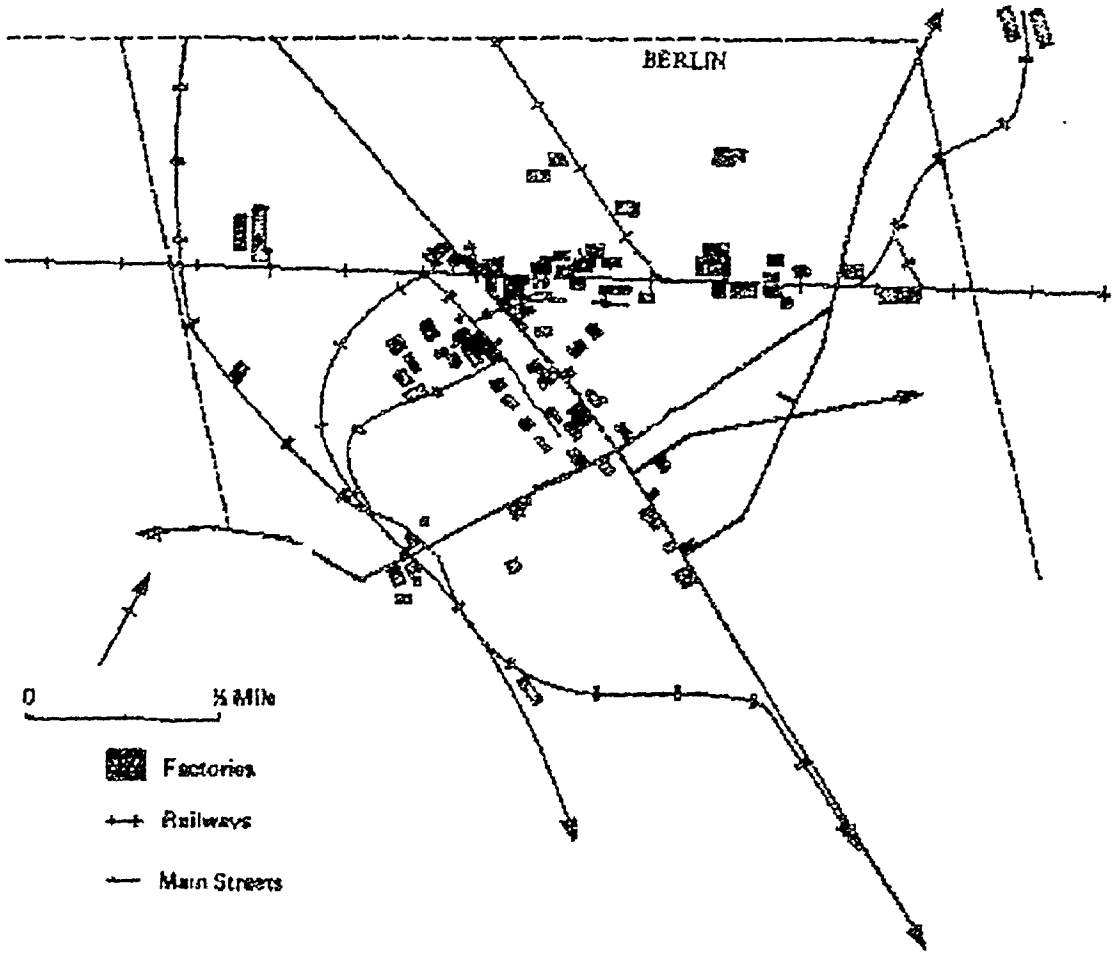


Figure 5-16. Spatial pattern of manufacturing in Kitchener, 1913.
Adapted from Bloomfield (1987:32)

Age differences in mortality

One of the most notable facets of the 1918 pandemic was the way it struck so heavily among the young adult population, the 20 to 40 year olds. This was, as was shown in the previous chapter, also the case here in Kitchener. But was there any spatial component to this age-related mortality? In order to attempt to answer this the population of victims was subdivided by age and then mapped (Figures 5-17 to 5-19). From these it can be appreciated that no particularly noticeable spatial variation exists. The 20 to 40 year old age group is most significant in all the wards. The younger group (0 to 19 year olds) also appears to be spread throughout the wards in the same proportions as the total population (Figure 5-20). The older group of victims, aged 41 to 90, is not as clear as the number of cases is rather small.

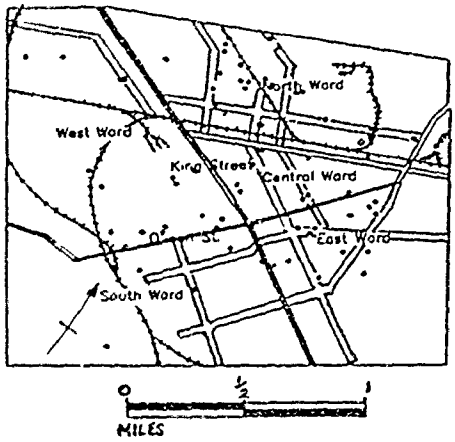


Figure 5-17. Influenza victims in the 20 to 40 age group.

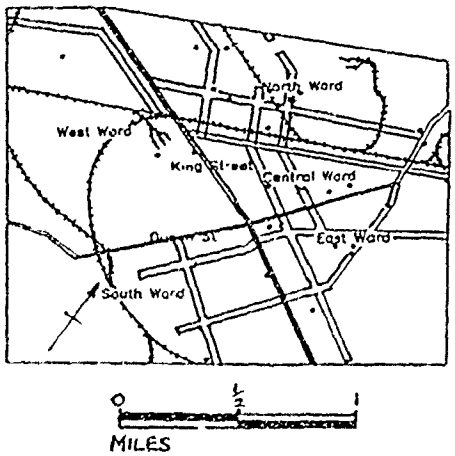


Figure 5-18. Influenza victims in the 0 to 19 age group.

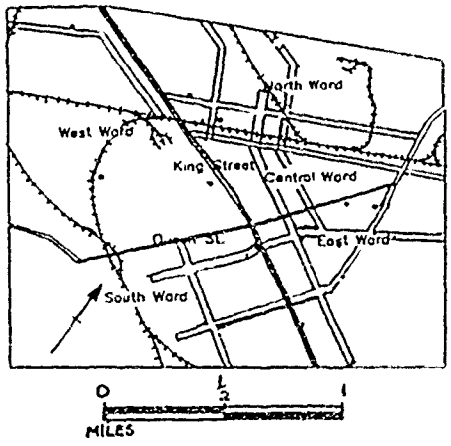


Figure 5-19. Influenza victims in the 41 to 90 age group.

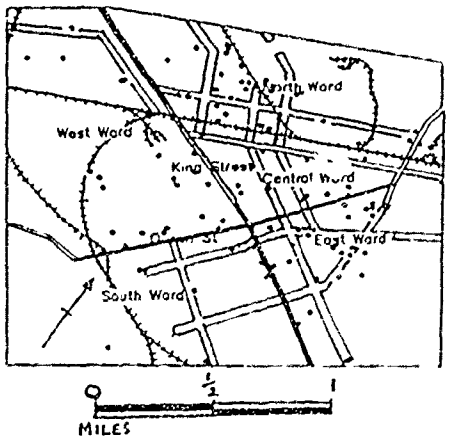


Figure 5-20. All influenza victims.

But were the victims in this age group similar to those who also died? Did they share the same socio-economic characteristics, etc. as their fellow victims? From the examination of the data (Table 5-8) it would appear that this age group, known to be of considerable importance in the expression of influenza mortality in 1918, did not significantly differ from the total population struck down by the disease. The two groups died throughout the entire month, with the average date being very similar. There was a slightly greater proportion of males killed in the 20 to 40 age group than in the total. This is similar to the experience elsewhere, as was discussed in earlier chapters. This age group would have been at the peak of their earning potential and thus the fact that the values related to the levels of taxation assessed and property values assessed are somewhat higher than those of the total population of victims, is not particularly surprising. Thus while this age group dominates the mortality profile they are not socially or economically demonstrably different from the other victims of this pandemic.

	Total	20 to 40 age group
Average date of death	15.058	15.029
Sex (1 = female, 2 = male)	1.548	1.603
Total taxes		
Mean	58.627	67.608
Min.	3.57	5.36
Max.	596.23	596.23
Assessed value		
Mean	1441.134	1614.273
Min.	120.00	180.00
Max.	11125.00	11125.00

Table 5-8. Comparison of influenza victims, Kitchener 1918. - 20 to 40 age group against all victims.

Chapter 6. Implications and conclusions.

We all labour against our own cure,
for death is the cure of all diseases.
Sir Thomas Browne.

Kitchener, Ontario undoubtedly suffered greatly from the influenza pandemic of 1918. The city was rocked, as was the entire globe, by this scourge. Around the world an estimated billion people suffered with 20 to 50 million succumbing to the disease. Here in Canada estimates of 2 million ill and at least thirty thousand dead demonstrate its dreadful passage. Within this immense toll in Canada the nineteen thousand-odd population of Kitchener witnessed the deaths of 127 of their number.

Kitchener's experience

Through this work, despite the myriad problems with extant data, it has been demonstrated that the Kitchener experience of this pandemic mirrored that of the rest of the world, particularly that of other North American cities. The epidemic curve, produced in Chapter 4, shows the same temporal rise and fall of the disease encountered elsewhere. The month of October 1918 saw the second

wave of the 1918-19 pandemic strike many North American cities with the morbidity and mortality associated with the outbreak dominating the affairs of these cities for a period of two to three weeks before fading away.

The mortality associated with the 1918 pandemic was unusual in that its preferred victims were in the 20 to 40 year age group. Influenza typically claims those at the extremes of age, yet the 1918 pandemic saw the young to middle aged adult become the target of choice. This pattern, as has been shown here, was very much the case in Kitchener.

Early examination of the primary source material relating to Kitchener's struggle with the disease revealed that many of the victims were manufacturing workers, often in the city's rubber factories. This prompted questions related to the possibility of a socio-economic factor in the mortality expressed in this pandemic. Were those factory workers being targeted in abnormally high numbers? Was there something unusual happening here in Kitchener as theory, and practice, had suggested that influenza was oblivious to status, striking all those vulnerable and exposed to the virus with complete disregard for their social and economic well-being? It does appear that there is an association between socio-economic factors and influenza fatalities. It appears that many of those who died came from working class households, households where the

breadwinner was employed in industries such as manufacturing and construction. There is an apparent association with higher population density, likely to be a result of lower socio-economic status. However, it is difficult to quantify the role of these factors.

It is also difficult to ascertain how important behavioural factors could have been. Factors such as seasonality, mobility, journey to work, transport methods, activity space, the social interactions, the workplace routine, proximity of workers, etc. could all modify how the disease is spread throughout the community. Even in the compact city of Kitchener in 1918 the nature of social behaviour will have affected the passage of the disease.

By and large the model put forward here is biological. The disease spread until it ran out of susceptibles. However it is also apparent that this pattern is modified by a number of ecological factors. These include age, income, mobility and occupation.

Thus in many ways, indeed if not all, Kitchener's experience would appear to be a very typical one for a North American city. This is true in terms of the timing of the disease, the length of time the disease held sway, the mortality

experienced in terms of those killed and the apparent indifference of influenza to socio-economic variations.

Influenza diffusion

Recent research into influenza diffusion (Cliff et al. 1986, Pyle 1986, Selby 1982) suggests two components to the spread of the disease. That is, there is a hierarchical component and a contagious component. The Canadian experience of the 1918 pandemic, as examined here, suggests that influenza spread through the urban hierarchy then at the city and neighbourhood level displayed contagious diffusion. The illness relocates through the nation's urban hierarchy and then contagious diffusion, through person-to-person transmission, at the city or neighbourhood level assumes greater importance. The first, hierarchical relocation, is responsible for the dispersal of the disease across the country from larger to smaller centres largely via the transportation networks, particularly the railways, and the second, contagious diffusion, sees the illness spread through neighbourhoods, towns and cities.

Within Kitchener it has been established that the disease appears to have started its spread through the city from the vicinity of the station. From there it spread across the city, claiming victims in all parts of the city within weeks. Within a month all known fatalities had occurred We have seen the rate and

extent of its spread, reaching across the entire 2 mile diameter of the city in just over a month.

Implications and directions for future research

This work has contributed to the knowledge of the influenza pandemic of 1918 in several ways. Firstly, by compiling the existing literature and producing maps of the spread of the disease at the global and national scale and, more extensively, it has documented the passage of the disease in a particular community. Here it has been established that it was the pandemic of influenza that struck Kitchener in October 1918. The pattern of mortality of the disease has been examined temporally and spatially. Furthermore, this pattern with regard to age, sex and socio-economic factors has also been analysed.

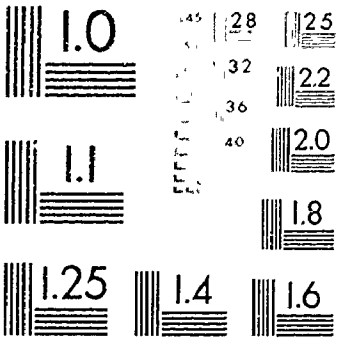
From this study there now exist a number of directions in which future researchers could proceed in gaining further insight into the pandemic of 1918. One of these relates to the impact of the housing environment upon health, as discussed in Chapter 5. Thus one possible direction, albeit a difficult one, could be detailed examination of the housing stock. This would be a painstaking and time-consuming task fraught with frustration as many of the dwellings have undoubtedly been demolished or changed beyond recognition.

Greater use of provincial records, such as the collated Medical Officer of Health reports, Department of Health reports and the papers of the Division of Public Health, is a direction that could be pursued. Another direction that could also provide further clues would be the detailed examination of church and school records, attendance or absenteeism rolls for schools and workplaces. Where these exist they could be a valuable source of morbidity data. Doctor's records would also be an immensely useful source, if they could be obtained. This kind of source material would enable us to develop a better understanding of the morbidity and the social impact of the disease.

From a geographical perspective the most valuable information that could be recovered would be that relating to population density, preferably at the household or block level. If, through any possible means, indications of the population density could be obtained this would open up the entire gamut of disease diffusion modelling and the examination of population potentials for the spread of disease. From these a greater understanding of the dynamics of disease diffusion could be gained.

3 of/de 3

PM-1 3½"x4" PHOTOGRAPHIC MICROCOPY TARGET
NBS 1010a ANSI/ISO #2 EQUIVALENT



Could it happen again?

Influenza has been one of the greatest killers throughout history, but it has done so without attracting the attention other plagues and diseases have. This is due, in part, to the relatively low case fatality rates. As an infectious disease it has potential to be a major killer. Even when it does not kill, as in the majority of cases, it exacts a vast cost medically, socially and economically. Humankind and influenza have a long history, the relationship between us and this "unvarying disease caused by a varying virus" (Kilbourne, 1980) should continue for many years to come as the virus continues to 'drift' and 'shift' to the frustration of those combatting it.

But are we likely to see a pandemic of the scale seen in 1918? We will continue to see influenza epidemics and pandemics. Influenza, through its perpetual evolution, re-emerges at epidemic levels every few years. Pandemics of influenza can also be expected to arise on occasion as the virus undergoes antigenic shift, keeping that one step ahead of the microbiologist.

However, some workers in this field are optimistic that we shall never again see the mortality that was visited upon the world in 1918 (Douglas 1987, Pettigrew 1983). Such proponents suggest that even if we endured another

pandemic on the scale of the 1918-19 outbreak with similar levels of morbidity it is unlikely that the mortality rates seen then would be repeated. This is thought to be valid if it is considered that the majority of deaths during that pandemic came from the secondary, bacterial, infections and not solely from influenza. Many of these secondary infections can be cured or contained using modern pharmaceuticals. It is felt that mortality may increase but it is likely to be among those groups that are typically vulnerable to influenza; the old, the young and the ill. Pettigrew (1983:137) sees the many social and medical developments of the intervening decades as playing a major role in reducing the impact of any future pandemic in Canada:

> If . . . we had another outbreak of the magnitude of the 1918 pandemic, how would Canada fare? In many ways, better. People in the most remote areas can communicate with others by radio telephone. There are more and larger hospitals, and social agencies to care for people without families. Federal and provincial health authorities would work together. Most important, we have available diagnostic tests and preventive vaccines, and powerful antibiotics for treating bacterial complications.
>
> Medically, then, we should have every confidence of a less terrifying and lethal situation. We can only hope that we would be as wealthy again in terms of human kindness.

This optimism is not shared by all, for example Crosby poses the question and can not find the same positive outlook:

> Can we have another killing pandemic of influenza? We don't really know what happened in 1918, and so we cannot justify optimism. A whole battery of antibiotics to protect the influenza patient against bacterial pneumonia has been developed . . . but the pneumonic complications of Spanish influenza may often have been only superficially bacterial, and indeed, in many cases were not complications at all, but simply the injury done to human lungs by a particularly virulent strain of virus.
>
> It is wiser to be humble than arrogant about influenza (Crosby, 1989:223).

The last great plague?

The claim that influenza is the 'last great plague', as Beveridge (1977) does, should be viewed with caution. Yes, influenza has the potential to again claim vast numbers and every year represents a major cost to our medical system and our society. However, there exist many other diseases that claim a massive toll, and many of these could be confronted and largely vanquished if the will was there. Gallagher (1969:41) claims that

> By virtue of its evasion of permanent immunization influenza has been termed 'the last of the unconquered scourges,' a label many public health people hold to be overblown. The WHO position is, no disease can be called 'the last unconquerable' while other abhorrences (such as onchocerciasis, blinding millions of Africans) still rage.

Lederberg adds that "our neglect of infectious disease in the poor majority of the world is not just a humanitarian disgrace; it leaves unchecked the seeds of our

parochial infection" (1991.31) as the diseases that evolve in these regions may reach out and strike us in the more developed countries.

Influenza continues to evolve

Thus the struggle between the human race and the influenza virus will, in all likelihood, continue. The host-parasite relationship will continue. Further the advances some see as beneficial to human health may not be of such value as

> [P]aradoxically, improvements in sanitation and vaccination leave the larger human herd more innocent of microbial experience, and may in the long run make us the more vulnerable. On the other hand, the loosening of ethnic barriers has made the human population a mite more variable, and in principle better equipped to deal with biological challenges. (Lederberg, 1991:31)

The influenza virus will continue to 'drift' and 'shift' and to cause human suffering. Fincher (1989:145) recorded that

> As evolutionary biologist Stephen Jay Gould recently told an AIDS-lecture audience" 'We've had a couple of generations of great fortune: since the . . . flu epidemic of 1918, there has not been a [lethal] pandemic disease that struck the human population. If you look through human history, a pandemic is everyday biology. With our usual hubris we felt that we'd learned through technological advances to be free of it forever. But we're not.'

So, the human race must be prepared for further assaults of influenza, with the possibility of massive morbidity and mortality.

Appendix I. All influenza deaths - Kitchener, October 1918.

No.	Date	Name
1	1	HILLER, Anna
2	2	WEY, Barbara
3	2	RUFF, Maria
4	3	MALINECK, John
5	4	OPPERMAN, Hilda
6	5	HAGEN, Otto
7	5	BUSCHERT, Lloyd
8	6	STALEY, Margaret
9	6	STRAUS, Lydia
10	6	PLANTZ, Martin
11	7	DOERR, Irene
12	7	LACKENBAUER, Gordon
13	7	NEDSIATKOWSKI, John
14	7	HELDMAN, Roy
15	7	WEBER, Fred C.
16	8	STEPPLER, Martha
17	8	GROTJOHN, Theodore
18	9	HELFERS, Fred
19	9	DOERR, Webourne
20	10	KRUPP, Robert
21	10	GROTJOHN, Ida
22	10	PLANTZ, William
23	10	LEHMAN, Annie
24	10	BULMER, Lelian
25	10	MOSER, Chas
26	10	MACKIE, George
27	10	FIGUERS, Florence
28	10	OLHEISER, Edward
29	10	HILLER, Ellen
30	11	KINZIE, Alice
31	11	STRONG, Webly
32	11	TROYAN, Franz
33	12	LEVANDOSKY, Viva
34	12	STRUB, Urban
35	12	MEISEL, Edward
36	12	BOLITSKY, Gertrude
37	12	REIKO, Milton
38	12	LUFT, Edna
39	12	STONER,Ruby
40	12	BOEHMER, Charlotte

No.	Date	Name
41	12	STEVENS, Art
42	13	KIRCHENBAUM, baby
43	13	KRAEMER, Lloyd
44	13	WEILER, Teckla
45	13	HUMMEL, Catherine
46	13	KIEFER, Ellen
47	13	VON NEUBRONN, Frank
48	13	HAGEN, Henry
49	13	BUCHER, Oscar
50	14	CISARCHUK, William
51	14	GEISEL, Louisa
52	14	HEIMBECKER, Kathleen
53	14	FEATHER, Elizabeth
54	14	KUNTZ, Edgar
55	14	DAVIDSON, Margaret
56	14	PAWSON, Clifford
57	14	KURSCHINSKI, Annie
58	14	LINDER, Cecil
59	14	RINGLE, Lily
60	14	HILTZ, Motilda
61	15	DAUB, Edward Eugene
62	15	SMITH, Cora
63	15	BOYER, Josephine
64	15	HATCH, Joshua
65	15	WOOD, Pearl
66	15	SMITH, Frank
67	15	COURTER, Mary
68	16	LEHMAN, Mary
69	16	FIGURES, Theresa
70	16	RESAW, Camelia
71	16	EMBRO, Joseph
72	16	KRAEMER, Harold
73	16	MARTIN, John
74	16	WINTERHALT, Annie
75	16	SHEPPERD, Floyd
76	16	ROTH, Albert
77	16	HEIMBECKER, Charles
78	16	OAKLEY, Oscar
79	17	RANDALL, Ida
80	17	KOSLAWSKI, Joseph
81	17	ECKSTEIN, Harold
82	17	SCHULTZ, Albert
83	17	KINGSLEY, Robert

No.	Date	Name
84	17	MOODY, William
85	17	CARTHEU, Fred
86	17	SCHLENDER, Leona
87	17	TUCKER, Alice
88	17	MITCHELL, James
89	17	FLORENCE, Gety
90	17	LINDNER, Harry K.
91	18	GREEN, Ida
92	18	SCHELL, Alfred
93	18	URSTADT, Nellie
94	18	WEIS, Edgar
95	18	ZUURHOUND, Elizabeth
96	18	HELM, Clara
97	18	SLUMKOFSKE, Emil
98	19	LINDER, Charles
99	19	COURTER, Rebecca
100	19	MORGAN, Ethel
101	19	SCHIPPANOWSKI, Mildred
102	19	QUIBELL, William
103	19	RICKERMAN, Nelson
104	19	LEMBKE, Margaret
105	19	BAST, Amos
106	20	KROETSCH, Alfred
107	20	SAUGEL, Walter
108	20	BADKE, Otto
109	20	KOT, Lucas
110	20	KRIGNER, Eileen
111	20	MICKUS, Harvey
112	21	ROHDENBERG, John
113	21	FORWELL, Sylvester
114	21	WILKINSON, Effie
115	22	SPIEGELBERG, Arthur
116	22	ODERBERG, Elsanora
117	22	KRAUSE, Charles
118	22	BANFELT, Ida
119	23	CRESMAN, Raymond
120	23	KUCHER, Walter
121	23	DAVIDSON, Elizabeth
122	23	BROWN, Arthur
123	24	BRYDEN, Fred C.
124	25	WITTAUOMSKI, Valeria
125	25	HINTZ, Norma
126	25	KUBE, Otto

No.	Date	Name
127	25	HEIMBECKER, Edward
128	28	KROHN, Augusta
129	29	HAMMER, Ida
130	31	BRAMM, Maud
131	31	LORENZ, Frederika

Appendix II. - Influenza deaths - Kitchener residents

No.	Date	Name	Age
1	1	HILLER, Anna	21
2	2	RUFF, Maria	22
3	3	MALINECK, John	37
4	4	OPPERMAN, Hilda	9
5	5	HAGEN, Otto	20
6	5	BUSCHERT, Lloyd	1
7	6	STALEY, Margaret	30
8	6	STRAUS, Lydia	51
9	6	PLANTZ, Martin	49
10	7	DOERR, Irene	28
11	7	LACKENBAUER, Gordon	3
12	7	NEDSIATKOWSKI, John	
13	7	HELDMAN, Roy	1
14	7	WEBER, Fred C.	39
15	8	STEPPLER, Martha	33
16	8	GROTJOHN, Theodore	26
17	9	HELFERS, Fred	26
18	9	DOERR, Webourne	28
19	10	KRUPP, Robert	25
20	10	GROTJOHN, Ida	25
21	10	PLANTZ, William	33
22	10	LEHMAN, Annie	22
23	10	BULMER, Lelian	25
24	10	MOSER, Chas	20
25	10	MACKIE, George	27
26	10	FIGUERS, Florence	14
27	10	OLHEISER, Edward	30
28	10	HILLER, Ellen	28
29	11	KINZIE, Alice	40
30	11	STRONG, Webly	37
31	11	TROYAN, Franz	89
32	12	LEVANDOSKY, Viva	35
33	12	STRUB, Urban	29
34	12	MEISEL, Edward	19
35	12	BOLITSKY, Gertrude	14
36	12	REIKO, Milton	4
37	12	LUFT, Edna	19
38	12	STONER,Ruby	1
39	12	BOEHMER, Charlotte	44
40	12	STEVENS, Art	27

41	13	KIRCHENBAUM, baby	0
42	13	KRAEMER, Lloyd	1
43	13	WEILER, Teckla	22
44	13	HUMMEL, Catherine	1
45	13	KIEFFIC, Ellen	28
46	13	VON NEUBRONN, Frank	25
47	13	HAGEN, Henry	30
48	13	BUCHER, Oscar	23
49	14	GEISEL, Louisa	25
50	14	HEIMBECKER, Kathleen	1
51	14	FEATHER, Elizabeth	55
52	14	KUNTZ, Edgar	17
53	14	DAVIDSON, Margaret	67
54	14	PAWSON, Clifford	31
55	14	KURSCHINSKI, Annie	21
56	14	LINDER, Cecil	13
57	14	RINGLE, Lily	29
58	14	HILTZ, Motilda	52
59	15	DAUB, Edward Eugene	1
60	15	SMITH, Cora	28
61	15	BOYER, Josephine	30
62	15	HATCH, Joshua	26
63	15	WOOD, Pearl	26
64	15	SMITH, Frank	36
65	15	COURTER, Mary	46
66	16	LEHMAN, Mary	19
67	16	FIGUERS, Theresa	25
68	16	RESAW, Camelia	70
69	16	EMBRO, Joseph	32
70	16	KRAEMER, Harold	2
71	16	MARTIN, John	75
72	16	WINTERHALT, Annie	37
73	16	SHEPPERD, Floyd	28
74	16	ROTH, Albert	25
75	16	HEIMBECKER, Charles	32
76	16	OAKLEY, Oscar	22
77	17	RANDALL, Ida	5
78	17	KOSLAWSKI, Joseph	25
79	17	ECKSTEIN, Harold	1
80	17	SCHULTZ, Albert	23
81	17	KINGSLEY, Robert	18
82	17	MOODY, William	33
83	17	CARTHEU, Fred	26
84	17	SCHLENDER, Leona	16

85	17	TUCKER, Alice	1
86	17	MITCHELL, James	26
87	17	FLORENCE, Gety	29
88	17	LINDNER, Harry K.	26
89	18	GREEN, Ida	28
90	18	SCHELL, Alfred	28
91	18	URSTADT, Nellie	25
92	18	WEIS, Edgar	31
93	18	ZUURHOUND, Elizabeth	16
94	18	SLUMKOFSKE, Emil	46
95	19	LINDER, Charles	10
96	19	COURTER, Rebecca	24
97	19	MORGAN, Ethel	9
98	19	SCHIPPANOWSKI, Mildred	5
99	19	QUIBELL, William	24
100	19	RICKERMAN, Nelson	28
101	19	LEMBKE, Margaret	
102	19	BAST, Amos	23
103	20	KROETSCH, Alfred	29
104	20	SAUGEL, Walter	28
105	20	BADKE, Otto	29
106	20	KOT, Lucas	45
107	20	KRIGNER, Eileen	2
108	20	MICKUS, Harvey	29
109	21	FORWELL, Sylvester	30
110	21	WILKINSON, Effie	29
111	22	SPIEGELBERG, Arthur	28
112	22	ODERBERG, Elsanora	27
113	22	KRAUSE, Charles	24
114	22	BANFELT, Ida	4
115	23	CRESMAN, Raymond	2
116	23	KUCHER, Walter	30
117	23	DAVIDSON, Elizabeth	40
118	23	BROWN, Arthur	34
119	24	BRYDEN, Fred C.	35
120	25	WITTAUOMSKI, Valeria	1
121	25	HINTZ, Norma	5
122	25	KUBE, Otto	45
123	25	HEIMBECKER, Edward	39
124	28	KROHN, Augusta	28
125	29	HAMMER, Ida	34
126	31	BRAMM, Maud	23
127	31	LORENZ, Frederika	80

Appendix III - Influenza victims by address

No.	Date	Name	Age	Address
1	1	HILLER, Anna	21	192 Waterloo St
2	2	RUFF, Maria	22	122-124 Weber
3	3	MALINECK, John	37	122-124 Weber
4	4	OPPERMAN, Hilda	9	17 Gordon Avenue
5	5	BUSCHERT, Lloyd	1	94 Lancaster St
6	5	HAGEN, Otto	20	34 Shoemaker Ave
7	6	PLANTZ, Martin	49	39 Krug
8	6	STALEY, Margaret	30	37 Pinke
9	6	STRAUS, Lydia	51	52 Louisa
10	7	DOERR, Irene	28	143 Weber W.
11	7	LACKENBAUER, Gordon	3	140 Weber W.
12	7	WEBER, Fred C.	39	155 Margaret Ave
13	8	GROTJOHN, Theodore	26	117 Walter
14	8	STEPPLER, Martha	33	4 David
15	9	DOERR, Webourne	28	143 Weber W.
16	9	HELFERS, Fred	26	84 Krug
17	10	FIGUERS, Florence	14	613 King St W
18	10	GROTJOHN, Ida	25	117 Walter
19	10	HILLER, Ellen	28	10 Stahle Ave
20	10	KRUPP, Robert	25	127 Waterloo
21	10	LEHMAN, Annie	22	100 Shanley St
22	10	MACKIE, George	27	11 Hilda Ave
23	10	MOSER, Chas	20	10 Ellen St W
24	10	PLANTZ, William	33	72 Queen St S
25	11	KINZIE, Alice	40	150 Edward St
26	11	STRONG, Webly	37	25 College St
27	11	TROYAN, Franz	89	46 Waterloo St
28	12	LEVANDOSKY, Viva	35	169 Joseph St
29	12	LUFT, Edna	19	28 Spring
30	12	REIKO, Milton	4	? Indiana St
31	12	STEVENS, Art	27	147 Lancaster St W
32	12	STONER,Ruby	1	36 Chapel
33	12	STRUB, Urban	29	22 Young St
34	13	BUCHER, Oscar	23	36 Elgin
35	13	HAGEN, Henry	30	190 Victoria St
36	13	HUMMEL, Catherine	1	16 Hermie Place
37	13	KIEFER, Ellen	28	133 Frederik St
38	13	KIRCHENBAUM, baby	0	265 Joseph St
39	13	KRAEMER, Lloyd	1	90 Glasgow St
40	13	VON NEUBRONN, Frank	25	105 Frederik St

No.	Date	Name	Age	Address
41	13	WEILER, Teckla	22	10 Spetz
42	14	GEISEL, Louisa	25	188 West Ahrens St
43	14	HEIMBECKER, Kathleen	1	12[illegible] Shanley St
44	14	HILTZ, Motilda	52	163 Lancaster St E
45	14	KURSCHINSKI, Annie	21	204 Waterloo
46	14	PAWSON, Clifford	31	125 King St E
47	14	RINGLE, Lily	29	93 Louisa St
48	15	COURTER, Mary	46	45 Mansion St
49	15	DAUB, Edward Eugene	1	2 Grove
50	15	SMITH, Cora	2	Suddaby School, Lancaster E
51	15	SMITH, Frank	36	50 Church St
52	15	WOOD, Pearl	26	193 Wilmot St
53	16	EMBRO, Joseph	32	114 King St E
54	16	FIGUERS, Theresa	25	613 King St W
55	16	HEIMBECKER, Charles	32	129 David St
56	16	KRAEMER, Harold	2	90 Glasgow St
57	16	LEHMAN, Mary	19	100 Shanley St
58	16	MARTIN, John	75	47 Henry
59	16	OAKLEY, Oscar	22	56 Braun St
60	16	RESAW, Camelia	70	15 Pandora
61	16	SHEPPERD, Floyd	28	60 W St. Leger
62	16	WINTERHALT, Annie	37	128 Weber E
63	17	CARTHEW, Fred	26	57 Chapel
64	17	ECKSTEIN, Harold	1 9	Foreman Place
65	17	KINGSLEY, Robert	[illegible]3	22 Margaret
66	17	KOSLAWSKI, Joseph	25	32 Bismark Ave
67	17	LINDNER, Harry K.	26	183 Ahrens St W
68	17	MOODY, William	33	74 Lancaster
69	17	SCHLENDER, Leona	16	42 Hohner
70	17	SCHULTZ, Albert	23	139 Edward
71	17	TUCKER, Alice	1	196 Waterloo
72	18	SCHELL, Alfred	28	87 Lancaster W
73	18	SLUMKOFSKE, Emil	46	47 Ellen St W
74	18	URSTADT, Nellie	25	2 Hilda Ave
75	18	WEIS, Edgar	31	15 E. Ellen St
76	18	ZUURHOUND, Elizabeth	16	79 Yonge St
77	19	BAST, Amos	23	39 Mansion St
78	19	COURTER, Rebecca	24	45 Mansion St
79	19	LEMBKE, Margaret	0	166 Elgin
80	19	LINDER, Charles	10	183 Ahrens St W
81	19	MORGAN, Ethel	9	153 King St E
82	19	QUIBELL, William	24	American Hotel, 5-7 Queen N
83	19	RICKERMAN, Nelson	28	8 Dill

No.	Date	Name	Age	Address
84	19	SCHIPPANOWSKI, Mildred	5	289 Courtland Ave E
85	20	BADKE, Otto	29	83 Water St N
86	20	KOT, Lucas	45	68 Charles St
87	20	KRIGNER, Eileen	2	16 Pinke St
88	20	KROETSCH, Alfred	29	106 Wellington St
89	20	SAUGEL, Walter	28	163 Joseph
90	21	FORWELL, Sylvester	30	98 Charles St
91	21	WILKINSON, Effie	29	30 Hohner Ave
92	22	BANFELT, Ida	4	Local orphanage, 461 King W
93	22	ODERBERG, Elsanora	27	118 Courtland Ave
94	22	SPIEGELBERG, Arthur	28	18 Pinke St
95	23	BROWN, Arthur	34	15 Ahrens St
96	23	DAVIDSON, Elizabeth	40	26 Ahrens St
97	23	KUCHER, Walter	30	7 Henry St
98	24	BRYDEN, Fred C.	35	49 Foundry
99	25	HEIMBECKER, Edward	39	315 Wellington St
100	25	KUBE, Otto	45	53 King St E
101	28	KROHN, Augusta	28	56 Mill St
102	29	HAMMER, Ida	34	147 Glasgow
103	31	BRAMM, Maud	23	204 Queen St S
104	31	LORENZ, Frederika	80	42 Mt Hope

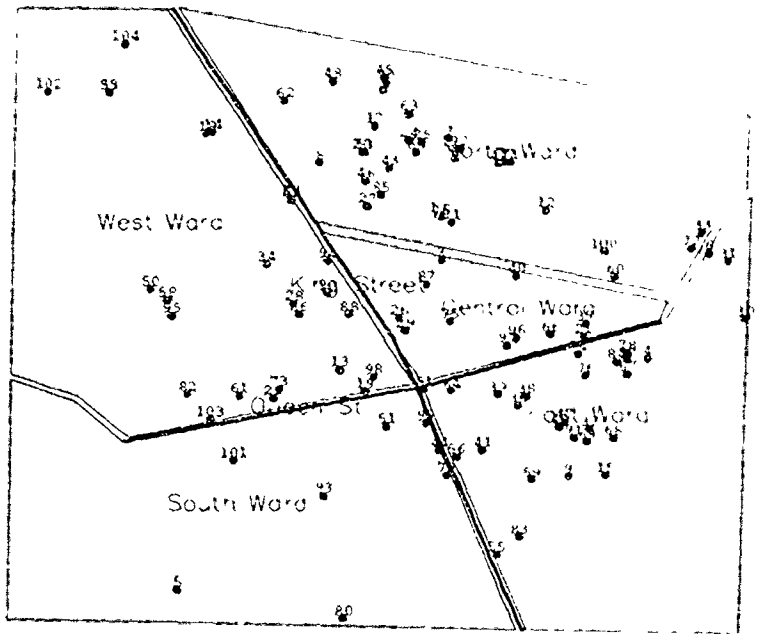


Figure App III-1. Influenza victims, Kitchener 1918 by case number.

Appendix IV - Influenza victims - occupation data

No.	Date	Name	Age	Occupation/Employer (or parent's/spouse's)
1	1	HILLER, Anna	21	Lang Shirt Co.
2	2	RUFF, Maria	22	Station Hotel
3	3	MALINECK, John	37	Barman - Station Hotel
4	4	OPPERMAN, Hilda	9	Father - salesman
5	5	BUSCHERT, Lloyd	1	Father - deliveryman
6	5	HAGEN, Otto	20	Merchants' Rubber
7	6	PLANTZ, Martin	49	'Gentleman'
8	6	STALEY, Margaret	30	Husband - agent
9	6	STRAUS, Lydia	51	Husband - Kaufmann Rubber
10	7	DOERR, Irene	28	Husband - accountant
11	7	LACKENBAUER, Gordon	3	Finisher (furniture)
12	7	WEBER, Fred C.	39	Anthes Furniture
13	8	GROTJOHN, Theodore	26	Labourer
14	8	STEPPLER, Martha	33	Husband - Dominion Tire
15	9	DOERR, Webourne	28	Accountant
16	10	FIGUERS, Florence	14	Father - bank messenger
17	10	GROTJOHN, Ida	25	Husband - labourer
18	10	HILLER, Ellen	28	Husband - rubber worker
19	10	KRUPP, Robert	25	Teamster
20	10	LEHMAN, Annie	22	Husband - labourer
21	10	MACKIE, George	27	HydroPower
22	10	MOSER, Chas	20	Consolidated Felt
23	10	PLANTZ, William	33	Merchants' Rubber
24	11	KINZIE, Alice	40	Husband - engineer
25	11	STRONG, Webly	37	Taxi service
26	12	LUFT, Edna	19	Star Whitewear Mfg.
27	12	REIKO, Milton	4	Father - cement worker
28	12	STONER,Ruby	1	Father - printer, News Record
29	12	STRUB, Urban	29	Printer, News Record
30	13	BUCHER, Oscar	23	Salesman
31	13	HAGEN, Henry	30	Merchants' Rubber
32	13	HUMMEL, Catherine	1	Father - Kaufmann Rubber
33	13	KIEFER, Ellen	28	Husband - bookkeeper
34	13	KRAEMER, Lloyd	1	Father - carpenter
35	13	VON NEUBRONN, Frank	25	Star Whitewear Mfg
36	13	WEILER, Teckla	22	Merchants' Rubber
37	14	HEIMBECKER, Kathleen	1	Husband - engineer
38	14	HILTZ, Motilda	52	Husband - marble worker

No.	Date	Name	Age	Occupation/employer
39	14	KURSCHINSKI, Annie	21	Kaufmann Rubber
40	14	PAWSON, Clifford	31	Soldier
41	14	RINGLE, Lily	29	Husband - Merchants' Rubber
42	15	DAUB, Edward Eugene	1	Father - engineer, Lion Brewery
43	15	SMITH, Cora	2	Teacher
44	15	SMITH, Frank	36	Agent
45	15	WOOD, Pearl	26	Husband - draftsman, Dominion
46	16	EMBRO, Joseph	32	Store-owner, merchant
47	16	FIGUERS, Theresa	25	Father - bank messenger
48	16	HEIMBECKER, Charles	32	Cabinet maker
49	16	KRAEMER, Harold	2	Carpenter
50	16	LEHMAN, Mary	19	Father - labourer
51	16	MARTIN, John	75	Soldier
52	16	OAKLEY, Oscar	22	Teamster, truck driver
53	16	SHEPPERD, Floyd	28	Inspector, public schools
54	16	WINTERHALT, Annie	37	Husband - cigar manufacturer
55	17	ECKSTEIN, Harold	19	Father - conductor
56	17	KINGSLEY, Robert	18	Dominion Tire
57	17	KOSLAWSKI, Joseph	25	Labourer
58	17	LINDNER, Harry K.	26	Consolidated Felt
59	17	MOODY, William	33	Mail carrier
60	17	SCHULTZ, Albert	23	Merchants' Rubber
61	17	TUCKER, Alice	1	Father - Lippert Furniture
62	18	SCHELL, Alfred	28	Merchants' Rubber
63	18	SLUMKOFSKE, Emil	46	Carpenter
64	18	URSTADT, Nellie	25	Husband - 'traveller'
65	18	WEIS, Edgar	31	Salesman/clerk
66	18	ZUURHOUND, Elizabeth	16	Student
67	19	BAST, Amos	23	Dominion Rubber
68	19	COURTER, Rebecca	24	Clerk, Agnew's Shoes
69	19	MORGAN, Ethel	9	Father - painter
70	19	QUIBELL, William	24	Merchants' Printing
71	19	RICKERMAN, Nelson	28	Carpenter
72	19	SCHIPPANOWSKI, Mildred	5	Father - Kaufmann Rubber
73	20	BADKE, Otto	29	Clerk - Faber's Grocery
74	20	KOT, Lucas	45	Dominion Tire
75	20	KRIGNER, Eileen	2	Father - teamster
76	20	KROETSCH, Alfred	29	Clerk - Fennell's Hardware
77	20	SAUGEL, Walter	28	Heller's Jewellery Store
78	21	FORWELL, Sylvester	30	Moulder, P. Gire's foundry
79	21	WILKINSON, Effie	29	Husband - Treas. John Forsyth
80	22	SPIEGELBERG, Arthur	28	Tannery worker
81	23	BROWN, Arthur	34	Salesman - Greb Shoe Co.

No.	Date	Name	Age	Occupation/employer
82	23	DAVIDSON, Elizabeth	40	Husband - tannery worker
83	24	BRYDEN, Fred C.	35	Dominion Tire
84	25	HEIMBECKER, Edward	39	Foreman
85	25	KUBE, Otto	45	Stableman
86	28	KROHN, Augusta	28	Husband - labourer
87	29	HAMMER, Ida	34	Husband - Dominion Tire
88	31	BRAMM, Maud	23	Husband - Merchants' Rubber

Appendix V. Doctors in Kitchener, October 1918.

Name

Algie, James (Queen Street Asylum)
Carmichael, Duncan Angus (Freeport Sanatorium)
Clive, Leonard Franklin (Kitchener - jail surgeon)
Faulds, R.W.
Hagmeier, John Edwin
Hagmeier, Louis Gordon
Hett, John Emil
Honsberger, Jerome Fry (local Medical Officer)
Honsberger, Henry H.
Kalbfleisch, Frederick Henry
Kirby, Thomas Sylvester
Lackner, Henry George
Lackner, Harry Mackie
McGillawee, John A. (Medical Health Officer)
Proctor, Arthur Douglas
Schnarr, Robert Werner
Wallace, William Thomas

Appendix VI. Isochrone map details.

The isoline map (Figure 5-10) was constructed using the Surfer software package. The initial point data was extracted from Mapmaker which had been used to construct the dot maps. The data extracted consisted of the x and y co-ordinates and a z variable consisting of the integer values denoting the 4 day period in which each fatality had occurred. This data was input into Surfer and a grid (.grd) file created.

This grid file was then passed into the TOPO portion of the Surfer package to produce a contour surface. Largely the default settings were utilised with the following exceptions.

Firstly the contour lines were set such as each 4 day period (each z value) would be a distinct contour. Thus the settings were:

Min contour:	1
Max. contour:	8
Contour interval:	1

One result of this is that the earlier periods, 1-3, rarely appear as there are relatively few cases from which to interpolate contours.

From the CONLINE option in the TOPO menu the following settings were altered to produce this image (Figure 5-10).

Conlab sub-menu	
Label format:	General
No. decimal digits:	0
Label height:	1
Contour label color:	1
Label curve tolerance:	1.015
Label to label distance:	0.01
Label to edge distance:	0.1
Smooth sub-menu	
Curve smooth?	Yes
Tension factor:	2

Furthermore the border marks were turned off so as to simplify the presentation. The file was then saved into HPGL format and imported into the Ventura desktop publisher document.

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